MATERNAL AND FETAL OUTCOME IN ANAEMIA COMPLICATING PREGNANCY

- A study in North Chennai.

Dissertation Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

in partial fulfilment of the regulations

for the award of the degree of

M.D. (O.G.) BRANCH – II
OBSTETRICS AND GYNAECOLOGY

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, INDIA.

FEBRUARY 2006

CERTIFICATE

This is to certify that the work presented in this dissertation in partial fulfilment of the degree **M.D.(Branch II) Obstetrics and Gynaecology** examination of the Tamil Nadu Dr. M.G.R. Medical University entitled "**MATERNAL AND FETAL OUTCOME IN ANAEMIA COMPLICATING PREGNANCY - A study in North Chennai"** is the bonafide work of **Dr. Florence Soumya Jacob**, Post-graduate student in M.D. (OG).

It was carried out and prepared under the over all guidance and supervision in the Dept. of Obstetrics and Gynaecology, Govt. Raja Sir Ramaswamy Mudaliar Lying-In Hospital, Chennai-600 013.

DEAN
Govt. Stanley Medical College &
Hospital,
Chennai-600 001.

Prof. DEVAMBIGAI, M.D. D.G.O.
SUPERINTENDENT
Dept. of Obstetrics and Gynaecology,
Govt. Raja Sir Ramaswamy Mudaliar
Lying-In Hospital,
Chennai-600 013.

DECLARATION

I, **Dr. FLORENCE SOUMYA JACOB** solemnly declare that this dissertation titled, **"MATERNAL AND FETAL OUTCOME IN ANAEMIA COMPLICATING PREGNANCY - A study in North Chennai"** is a bonafide work done by me at Govt. RSRM Lying-in Hospital and Govt. Stanley Medical College during 2004-2005 under the guidance and supervision of my Superintendent **Prof. DEVAMBIGAI, M.D., D.G.O.**

The dissertation is submitted to The Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of **M.D. Degree (Branch – II) in Obstetrics and Gynaecology.**

Place : Chennai.

Date :

(Dr. FLORENCE SOUMYA JACOB)

CONTENTS

ACKNOWLEDGEMENT

I am extremely grateful to the Dean of Stanley Medical College, **Dr. T. RAVEENDRAN, M.D., D.T.C.D.,** for permitting me to use the hospital material for my study.

I owe a deep sense of gratitude to professor **Dr. Devambigai, M.D., D.G.O.,** Superintendent, Govt. Raja Sir Ramaswamy Mudaliar Lying-In Hospital, Chennai-600 013 for her valuable advice, guidance and help in doing this study.

I am deeply indebted and grateful to my Professor and guide **Dr. Latha, M.D.,D.G.O.** for her valuable guidance.

I am also very grateful to my Professors **Dr. Anjalakshi, M.D., D.G.O., Dr. Prema, M.D., D.G.O. Dr. Roopa, M.D., D.G.O.** and Assistant Professors for their valuable support.

Last but not the least, my sincere thanks to all the patients who cooperated for this study, without whom this study could not have been possible.

KEY TO MASTER CHART

1) LSCS. : Lower segment caesarean section

2) L-Naturale : Labour Naturale

3) PPH : Post Partum Haemorrhage

4) HbsAg : Hepatitis B Antigen

5) LRI : Lower Respiratory Infection

6) UTI : Urinary Tract Infection

INTRODUCTION

Anaemia is a major public health problem especially among the poorer segments of the population in developing countries like India and it is one of the major challenges an obstetrician faces in his / her carrier.

In India, it is frequently severe and contributes to maternal mortality and reproductive health morbidity. It deserves more attention than its currently receiving. Recently lot of programmes have been focussed on safe motherhood but maternal anaemia remains a problem of great concern.

Gender discrimination is another important factor in India as the girl child, right from youth, is denied proper food and education.

Anaemia in pregnancy accounts for 25 percent of deaths due to associated causes and 11.5% of all maternal deaths (Mudaliar). Anaemia contributes to 10-15% of direct maternal deaths in India. (Mudaliar)

An estimated 60% of all pregnant women in developing countries have anaemia. Anaemia either directly or indirectly contributes to about 20% of maternal deaths in third world countries.

Among all causes of anaemia, nutritional anaemia is of greatest concern, 90% due to iron deficiency (Fenton 1997).

In places where malaria or hook worm infestation is endemic, prevalence of anaemia is as high as 90% (Agarwal AK et al 1999). Konnon

(1994) studied the incidence of anaemia in adolescent girls in slum areas in the city of Baroda, 98% of adolescent girls were anaemic. This adversely affects reproductive performance.

Multipara, multiple pregnancy, blood donors and persons with a diet low in meat / vegetarian (Chegget et al 1996) are more prone for anaemia. Adolescents because of their low body iron stores are also at risk.

In India the prevalence of iron deficiency anaemia has come down due to fortification, prophylactic iron supplementation, better health care programmes aimed at women and children (School Hedige 1994).

The incidence and prevalence of anaemia in the pregnant women of North Chennai is very high. Most of the women attending the antenatal OPD have a Hb around 8-10gm/dl. These patients come from a low socioeconomic strata. They are unable to come for regular antenatal checkups. They prefer to work and earn their livelihood rather than get admitted and get their anaemia treated. This dissertation attempts to analyse the maternal and fetal outcome of pregnancy in anaemic women and study the effect of birth spacing in improving the same.

Prevention is better than cure. This study could guide us as to the levels at which we need to direct our preventive measures to check the progress of anaemia in antenatal women and improve our out reach services thereby identifying anaemia in the adolescent stage.

NEED FOR THE STUDY

Prevention of anaemia is essential in normal care to have a healthy baby from a healthy mother and thereby build a healthy nation.

Anaemia is the most important complication in pregnancy in developing countries not only because of its high incidence but also because of its severity. WHO UNICEF collaboration survey in developing countries and ICMR studies in Indian context revealed that out of the total women suffering from anaemia 2/3 are pregnant and lactating mothers (Nailk D. Jayshree & Malatikeshan – 1992).

Anaemia of pregnancy is mainly nutritional-iron, folate and B12 deficiency; most commonly due to non-availability of correct food and food taboos and cooking customs. Studies reveal that socioeconomic, cultural factors influence dietary inadequacy during pregnancy which is attributed to poor purchasing power, illiteracy, ignorance regarding nutritive value of readily available cheaper foodstuffs, cultural taboos, superstition and large family (Menon Krishnan et al 1995, Rull Benett and Brownlinding 1993, Nailk / D Jayashree 1992). Factors like chronic illness, haemorrhage short birth intervals, parity will also influence the prevalence of anaemia during pregnancy (Ratnan et al 1993). Severe forms of anaemia in III trimester of pregnancy are invariably associated with cardiac failure, 20% deaths, low birth weight, prematurity, perinatal and infant mortality (Mennon Krishnan et al 1996).

According to Hassan Masood 1991 anaemia is prevalent in 50-90% pregnant women in India which is No.1 Killer.

Despite considerable improvement and awareness in Antenatal care in developing countries and inspite of MCH programmes and National Anaemia Prophylaxis in India, anaemia remains a great concern with regard to maternal morbidity and mortality and adverse outcome (Singh Krishnan et al 1995).

Anaemia may antedate conception, its often aggravated by pregnancy and the accidents of labour may perpetuate it. It is one of the prime concerns of the antenatal care to forestall it, for the safety of labour and puerperal state. The incidence of anaemia has been on the rise for past several decades in different parts of the world, more so in developing countries. Recent reports indicate the increase in maternal morbidity, mortality and adverse fetal outcome when anaemia complicates pregnancy. To decrease this, obstetricians have been stressing on regular antenatal checkup. Thus an effort has been made to know the effects of anaemia on fetomaternal outcome in this study.

AIM OF THE STUDY

To study women presenting in the antenatal department with various degrees of anaemia and find out its effect on

- Maternal morbidity and mortality
- Mother in puerperium

- Fetal morbidity and mortality
- Baby in the neonatal period

HISTORICAL ASPECTS OF ANAEMIA

The work of Hedin and Wintrobe in assessing the volume of packed red cells by various types of hematocrit and the work of Keith, Rewntree and Garathy in estimating blood volume led to the accurate laboratory definition of the presence or absence of anaemia.

Pierre Blaud in 1832, discovered that ferrous sulphate tablets were effective therapy for iron deficiency anaemia.

In 1919 Sir William Osler classified anaemia as occurring during pregnancy, following post partum haemorrhage, associated with post partum sepsis or other postpartum anaemia.

In 1922 Price quantitated the variation in red cell size seen in various types of anaemia.

In 1932 Wintrobe devised the concept of red cell indices.

REVIEW OF LITERATURE

ANAEMIA IN PREGNANCY

Anaemia is perhaps the most common complication in pregnancy met with in the tropics.

Studies carried out in the forties and fifties had shown that in India.

- In India the incidence of anaemia is 40% – 90%
- Iron and folic acid deficiency were the major factors responsible for anaemia, due to nutritional deficiency (diet).
- Anaemia in pregnancy accounts for 25% of deaths due to associated causes and 11.5% of all maternal deaths.(Mudaliar)
- Anaemia in pregnancy was associated with a high perinatal loss.

Management of anaemia in pregnancy was therefore accorded a very high priority both in obstetric and public health practice. The National Anaemia Prophylaxis Programme was implemented in 1972 hoping to reduce its prevalence and severity in pregnancy. But unfortunately studies carried out during the late eighties in India failed to show any change in either the magnitude or its adverse obstetric consequences.

DEFINITION

A clear definition of anaemia in women is complicated by normal variations of haemoglobin levels between men and women, between pregnant and non-pregnant, between the various socio-economic standards and between those who receive nutritious diet and iron supplements and those who do not.

In anaemia there is a low haemoglobin concentration resulting in decreased oxygen carrying capacity of the blood.

The WHO expert group had defined anaemia as a haemoglobin level less than 11g/dl. The Centers for Disease Control (1990) has defined anaemia as less than 11g/dl in the first and third trimesters and less than 10.5g/dl in the second trimester.

In India anaemia is defined as Hb < 10gm /dl as given by Federation of Obstetrics and Gynaecological Society of India (Mudaliar).

This difference in the cut-off value according to gestational age is based on the fact that the fall in haemoglobin level in pregnancy is most marked in the mid –trimester due to a relatively greater expansion of the plasma volume compared with the haemoglobin mass and red cell volume. Late in pregnancy, plasma expansion essentially ceases while haemoglobin mass continues to increase.

Degrees of Anaemia

Asian Journal of OG - 1999

Mild Anaemia	9.1-11gm/dL
Moderate	7.1 – 9gm/dL

Severe Hb $\leq$ 7gm/dL

ETIOLOGY OF ANAEMIA IN PREGNANCY

Physiological anaemia of pregnancy.

Pathological

- **Deficiency anaemia (Isolated or combined)**
    - Iron deficiency
    - Folic acid deficiency
    - Vitamin B12 deficiency
    - Protein deficiency
- **Haemorrhagic**
    - Acute following bleeding in early months or APH
    - Chronic – Hookworm infestation, bleeding piles
- **Hereditary**
    - Thalassemias
    - Sickle cell haemoglobinopathies
    - Other haemoglobinopathies
    - Hereditary hemolytic anaemias.
- RBC membrane defects
- Spherocytosis
- Bone marrow insufficiency
    - Hypoplasia or aplasia due to irradiation
    - Drugs aspirin, indomethacin

- Anaemia of infection (Malaria, tuberculosis)
- Chronic renal disease or neoplasm.

ETIOLOGY OF ANAEMIA IN PREGNANCY

Changes in Hb levels during pregnancy:

In pregnancy there is progressive increase in circulating blood volume due to increase in plasma and RBC volume. Increase in blood volume ranges from 30-70% of the non-pregnant level. The increase in plasma volume is greater (40 to 50%) than the rise in RBC volume (18-25%) so there is a fall in Hb levels. The fall is physiological because :

a) It begins in the first trimester when iron needs are fully met.

b) It occurs in well nourished women also

c) It is not eliminated by administration of iron.

Repeated pregnancies at short intervals. It takes 2 years to replenish 1000mg of iron lost in pregnancy and lactation.

Folate deficiency is seen among lower socioeconomic groups (as vegetable intake especially green leafy vegetables, is very low, in mutipara and in malabsorption syndrome.

IRON REQUIREMENT IN PREGNANCY

In a typical singleton gestation, the maternal need for iron averages close to 800mg – 300mg for the fetus and placenta and 500mg, if available, for maternal haemoglobin mass expansion, 200mg are shed through gut, skin and urine. The total amount (1000mg) exceeds the iron stores of most women and results in iron deficiency anaemia.

Iron deficiency anaemia during pregnancy is the consequence primarily of expansion of plasma without normal expansion of maternal haemoglobin mass (Williams 22nd ed.)

IRON ABSORPTION IN PREGNANCY

Early in pregnancy iron absorption is decreased. It shows a marked improvement from about the twentieth week, reaching three times the control value by the thirty sixth week of gestation. Despite this, the iron requirements of pregnancy cannot be adequately fulfilled even from a diet of over 2000 kcal/day. The deficit of iron, towards the last trimester may be as high as 4-5mg / day.

In health, the **serum iron** concentration of adult non-pregnant women lies between 13 and 27 μmol/l. It shows marked individual diurnal variation and fluctuates even from hour to hour. **The total iron binding capacity (TIBC)**. In the non-pregnant state lies in the range 45-72 μmol/L. It is raised in association with iron deficiency and low in chronic inflammatory states. In the nonanaemic person the TIBC is approximately one third saturated with iron. Serum iron even in combination with TIBC is not a reliable indicator of iron stores because it fluctuates so widely, is affected by recent ingestion of iron and infection (De Swiet 4th edn).

Serum Ferritin is stable, not affected by recent ingestion of iron, reflects the iron stores. In iron deficiency, serum ferritin is the first to fall. Serum ferritin is estimated by sensitive immunoradiometric assay. Anaemic mothers have low serum cord ferritin.

Serum Transferin Receptor (TfR)

Is present in all cells as a transmembrane protein that binds transferrin iron and transports it to the cell interior. Serum TfR is a reliable indicator of cellular iron status. TfR will give a true reflection of tissue iron deficiency in pregnancy when ferritin may be low because of mobilisation and in chronic inflammatory disease when ferritin is inappropriately elevated because of release from cells.

Marrow Iron – No stainable iron (Haemosiderin) may be visible once serum ferritin has fallen to below 30μg/l (Deswict).

RDW – Red Cell Distribution Width. It is an index of the presence of heterogenous RBC with different diameters. If it is more than 15% it is abnormal. It occurs even before the peripheral smear becomes microcytic hypochromic.

FOLIC ACID DEFICIENCY

It was called pernicious anaemia of pregnancy. It is found in women who do not consume fresh green leafy vegetables, legumes or animal protein. Folic acid requirement is 400μg/day in the pregnant women (Williams's)

The earliest biochemical evidence is low plasma folic acid concentration. The earliest morphological evidence is hypersegmentation of neutrophils, and newly formed erythrocytes are macrocytic.

Additional folic acid is given in

1) Multifetal pregnancy

2) Hemolytic anaemia

3) Crohn’s disease

4) Alcoholism

5) Inflammatory skin disorders

6) Previous baby had neural tube defects

VITAMIN B12 DEFICIENCY

In addisonian pernicious anaemia, a lack of intrinsic factor results in failure to absorb vitamin B12. Infertility is a complication.

Vit. B12, deficiency is more likely following

1) Partial or total gastric resection

2) Crohn's disease

3) Ileal resection

4) Bacterial overgrowth in the small bowel.

ANAEMIA DUE TO BLOOD LOSS

Hookworm infestation is the most common cause in tropical countries

1) Ankylostoma duodenale – 0.25 ml/worm /day

2) Necator americanus 0.03 ml/worm/day

ANAEMIA DUE TO HEMOLYSIS

Falciparum malarial infections may cause hemolytic anaemia in addition to all the hemolytic anaemias of genetic origin.

Anaemia associated with infections

1) UTI

2) HIV perse does not cause anaemia, though in rare instances anaemia as a part of pancytopenia due to HIV infection have been reported.

Chronic renal disease results in decreased production of erythropoietin and hence anaemia.

CLINICAL FEATURES

They depend more on the degree of anaemia than anything else.

In majority, the patients have got no symptoms and the entity is detected accidentally during examination.

SYMPTOMS

Lassitude, a feeling of exhaustion may be the earliest manifestation.

Anorexia, indigestion

Palpitation caused by ectopic beats

Dyspnoea

Giddiness

Swelling of legs.

ON EXAMINATION

- Pallor of varying degrees
- Glossitis, stomatitis
- Oedema legs due to hypoproteinemia or associated pre-eclampsia
- Soft systolic murmur
- Crepitations at the base of the lungs due to congestion.

COMPLICATIONS OF SEVERE ANAEMIA

During Pregnancy

The following complications are likely to increase

1) Pre-eclampsia

2) Intercurrent infection

3) Heart failure at 30-32 weeks of pregnancy

4) Preterm labour

During Labour

1) Cardiac Failure : May be due to accelerated cardiac output which occurs during labour or immediately following delivery.

2) Shock

3) Post partum haemorrhage

4) Uterine inertia – it is not a common associate

During Puerperium

1) Puerperal sepsis

2) Subinvolution

3) Failing lactation

4) Peripheral venous thrombosis

5) Pulmonary embolism

Risk Periods

1) AT 30-32 wks of pregnancy

2) During labour

3) Immediately following delivery

4) In puerperium 7-10 days following delivery due to pulmonary embolism.

Anaemia directly or indirectly contributes to about 20% of maternal deaths in the third world countries.

Effects on the baby :

Increased incidence of

1) Low birth weight

2) Preterm babies

3) Intrauterine death – due to severe maternal anoxemia

Net effect

Increased perinatal loss

Treatment

This depends on the Hb level and the gestational age at which the patient presents.

All patients with Hb≤ 7gm/dl must be admitted.

Patients with Hb < 5gm/dl and especially those with Hb < 2.5gm/dl and PCV< 15% congestive failure is always present.

Congestive failure is a contraindication for parenteral iron therapy.

Nothing can be more dangerous than terminating pregnancy in a patient with severe anaemia.

Rest, sedation, digitalis and diuretics to control the failure and repeated packed cell transfusion to improve anaemia are useful in tiding over the crisis.

For patients who are not in failure

1) Parenteral iron therapy

Patients who

a. Require a quick response especially when she is near term

b. Cannot tolerate iron by mouth

c. Do not show satisfactory response to oral iron

The Hb increases by 0.7 to 1gm/100 ml per week.

A) Intravenous Route :

TDI – Total Dose Infusion

The deficit of iron is first calculated and the total amount of iron required to correct the deficit is administered by a single sitting IV infusion.

Iron Dextran is used.

Advantages :

1) Eliminates repeated, painful IM injections

2) Treatment is completed in a day

3) It is less costly

Limitations

1) As the maximum response does not appear before 4-9 weeks, the method is unsuitable if 4 weeks are not available for delivery. It is most suitable between 30-36 weeks of pregnancy.

2) Previous history of reaction to parenteral therapy.

3) IV iron can produce severe anaphylactoid reactions.

TDI = (Hb deficit x weight in Kg x 2.21) + 1000 in mg

B) Intramuscular Route :

Iron dextran (Imferon)

Iron sorbitol citric acid complex

(Jectofer)

Oral iron should be suspended 24 hours prior to parenteral therapy to avoid reactions.

Drawbacks :

1) Painful

2) Abscess formation and discolouration of skin over the injection site especially with imferon.

3) Pyrexia, lymphadenopathy, headache, nausea, vomiting and allergic reactions.

C) Blood / Packed cell transfusion

1) Correct anaemia due to APH or PPH

2) Severe anaemia beyond 36 weeks

3) Refractory anaemia

4) Associated infection

D) Oral Iron Therapy

Ferrous gluconate, ferrous fumarate or ferrous succinate.

Fersolate – 200mg ferrous sulphate which contains 60mg of elemental iron.

Adverse effects :

Most common – constipation

Epigastric pain, nausea, vomiting and diarrhoea are other effects.

The reticulocyte count increases by 7-10 days.

Hb. rises at the rate of 0.7gm/100ml per week.

To replenish iron stores oral therapy should be continued for 3 months after anaemia is corrected.

The National Anaemia Prophylaxis Programme distributes folifer tablets 100mg of elemental iron and 500μg of folic acid to all pregnant women who are not anaemic for at least last 100 days of pregnancy.

For patients with Hb between 7gm / dl and 11gm/dl – 2 tablets of folifer for 103 days.

Patients with Hb less than 7gm / dl are referred to the first referral centres.

All patients should have their motion screened for ova and cyst. Those with hookworm infestation are given Tab. Albendazole 400mg stat.

MANAGEMENT DURING LABOUR

First Stage :

The patient should be in bed and should lie in a position comfortable to her.

Arrangements for oxygen inhalation to be kept.

Strict asepsis to minimise puerperal infection.

Second Stage

Asepsis is maintained

Prophylactic outlet forceps or vacuum delivery to shorten the duration of second stage.

IV methergine 0.25mg at the time of delivery of the head.

Third Stage

Be very vigilant

Significant blood loss to be replaced but post partum overloading of heart should be avoided.

Puerperium

Prophylactic antibiotics to prevent infection.

Iron therapy for 3 months after delivery.

PARAMETERS USED IN THE DIAGNOSIS OF ANAEMIA

1. Hemoglobin concentration in g/dL 11g/dl (WHO)

2. Hematocrit or packed cell volume expressed as a percentage 35-45% (< 30% anaemia).

3. Red blood cell count in million per cubic millimetre (<4 million/mm^3)

4. Serum iron : normal : 60-150 μg/dL

5. Percentage saturation of transferrin – (Decreased in anaemia).

   Normally 1:3 saturated. Less than 15% saturation in Iron deficiency anaemia.

6. Total iron binding capacity (Increased in Anaemia)

   Normal : 45-72μmol/L

7. Serum Ferritin : Decreased in Anaemia

Normal 15-300 μg/L

8. Serum folate : Normal : 2-20 μg/L And

   Red Cell folate : Normal : > 100μg/L

   To diagnose folic acid deficiency

9. Peripheral blood smear to know the type of anaemia and look for malarial parasites.

INDICES USED IN DIAGNOSIS OF ANAEMIA

- **MEAN CORPUSCULAR HEMOGLOBIN (MCH)**

  $$\frac{\text{Hb in g per 1000 ml of blood}}{\text{RBC in million per cu. mm}}$$ In Picograms

  The normal value is 29.5 mico-micrograms.

- **MEAN CORPUSCULAR VOLUME (MCV)**

  $$\frac{\text{Volume of packed cells per 1000 ml}}{\text{RBC in million per cu. mm}}$$

  The normal value is 68 cubic microns

  > 90 => macrocytosis.

- **MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)**

  $$\frac{\text{Hb in g per 100 ml}}{\text{Volume of packed cells per 100ml}} \times 100$$

  It is normally 34%

  It represents the actual hemoglobin concentration in the blood and is the key to iron deficiency anaemia.

- **COLOUR INDEX**

  $$\frac{\text{Hb (Percentage of normal)}}{\text{RBC (Percentage of normal)}}$$

  It is normally one. If it is less then one the anaemia is hypochromic. If above one, it is hyperchromic.

MATERIALS AND METHODS

1000 antenatal patients attending the AN-OPD at the Govt. Raja Sir Ramaswamy Mudaliar Lying-in Hospital in the year 2004-2005 were selected for the study. These patients were screened for anaemia as is routinely done.

INCLUSION CRITERIA

- Patients of all three trimesters, with moderate or severe anaemia

EXCLUSION CRITERIA

Patients with low haemoglobin levels whose pregnancy ended in an abortion or turned out to be a molar pregnancy were excluded from the study. Patients with PIH were also excluded from the study.

For all the patients in the study the following parameters were measured

- Haemoglobin concentration in g/dl
- Haematocrit %
- Total RBC count million / mm^3
- Total WBC count
- Platelet Count
- Peripheral smear for type of anaemia and to rule out malaria

- MCV (Mean Corpuscular volume)
- MCH (Mean corpuscular haemoglobin)
- MCHC (Mean Corpuscular Haemoglobin Concentration)

METHODS OF ESTIMATION OF HAEMOGLOBIN

In Sahli's method, which is commonly used in laboratory practice, the haemoglobin is converted to acid haematin by placing 0.1 N HCI upto mark 10 in the graduated tube, and adding 20 cu.mm of blood to it. It is then diluted with water till the colour matches with the standard colour. It is better to take the reading in grams of haemoglobin per 100 ml rather than in percent Hb. If the reading is reported as per cent, how much Hb corresponds to 100% in the instrument must be indicated, as they may vary from 14 to 16gm in different instruments.

ESTIMATION OF RED CELLS

The concentration of RBC or WBC may be obtained by counting them directly after suitable dilution with special solutions. The instrument used for this purpose is called a' haemocytometer.

The diluting fluid that is usually used for RBC count is Hayem's fluid. It contains 0.5 gms. of sodium chloride and 2.5 gms. of sodium sulphate, which together give it isotonicity with blood. It also contains 0.25 gm. of mercury perchloride which along with the sodium sulphate also prevents clumping of the cells. Mercuric perchloride fixes the cells or makes the transparent cells translucent.

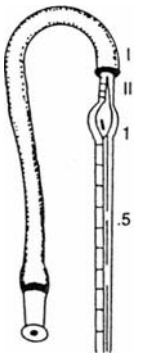
The method consists of spreading a film of suitably diluted blood on a special glass slide on which are marked squares of known dimensions. The area is divided into 16 or 25 large squares. Each large square is again divided into 16 small squares. Each small square is 1/20 mm at the edge. The area is therefore 1/400 sq. mm. The thickness of the film when the cover glass is in position is 0.1 *mm.* The film standing on 1 small square will be 1/4000 cu. mm. The stem and the pipette bulb are so arranged that a dilution of 1 : 200 is obtained. . The number of RBC in 80 small squares is counted. If N is the total number counted in all the 80 squares, the average number in each square is then N/80. The volume of fluid in each small square is 1/4000 cu. mm. The dilution is 1/200. Therefore the number of cells in 1 cu. mm of undiluted blood is N x 10,000. Thus, the value can be derived by adding 4 zeros to the total number obtained by counting the cells in 80 small squares.

For counting WBCs a different diluting fluid is used, containing acetic acid to destroy RBCs and methylene blue to colour the nuclei of WBCs and the dilution is 1/20. There are four large squares ($1mm^2$) at four corners out side the RBC counting chamber not shown in the figure. Each is divided into 16 smaller squares. The number of WBCs in all the four large squares are counted and value multiplied by 50 gives the WBCs in 1 cu.mm.

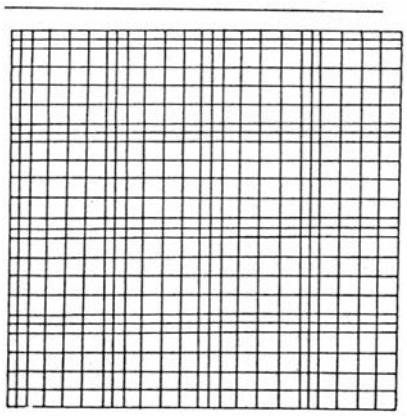
PERIPHERAL BLOOD SMEAR

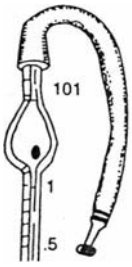
Make a blood smear on a glass slide, stain it with Leishman stain and examine it. With this, one can *(i)* get an idea of the changes in size, shape, colour of RBCs in anaemias. Abundant presence of small pale staining cells with variation in size (anisocytosis) and shape (poikilocytosis) suggest microcytic hypochromic anaemia. Macrocytosis is observed when cells are large in size and volume and seen in megaloblastic anaemia. Target cells seen in haemoglobinopathies are flat cells with haemoglobin arranged as a central mass and an outer ring with a pale zone in between. One can also detect the presence of malarial parasite if patient has malaria.

THOMA ZEISS COUNTING CHAMBER



WBC Pipette





RBC Pipette

HAEMOCYTOMETER

The patients were then followed upto delivery and the following parameters of perinatal outcome were noted.

- Alive or dead fetus at delivery
- Term or preterm delivery
- Mode of delivery – normal / instrumental / caesarean.
- Birth weight of the baby
- APGAR scoring at 1 minute and 5 minutes
- Any early neonatal complications or death.

The following parameters of Maternal Outcome were noted :

- Intercurrent infection
- Compensated or in congestive failure
- Shock
- PPH
- Puerperal sepsis
- Subinvolution
- Thromboembolism
- Maternal Death

OBSERVATION, RESULTS AND DISCUSSION

A total of 1000patients attending the antenatal OPD were taken at random into the study.

They were screened for anaemia.

There were a total of 137 patients who fell into the category of moderate and severe anaemia.

The maternal and fetal outcome in these 137 patients were studied in great detail.

Total number of patients = 1000

≤ 7gm /dl	=	25	Total 137
7.1 to 9gm/dl	=	112	
9.1 to 10gm/dl	=	786	
10.5 to 11 gm/dl	=	40	
> 11gm/dl	=	37	

13.7% belonged to moderate and severe anaemia. Maximum number of patients were mildly anaemic, 78.6% having Hb 9.1 to 10gm/dl.

70% of the population works in factories located in North Chennai and belong to the lower economic strata (Group V)

TABLE – 1

AGE / GRAVIDA
Patients with Hb ≤ 9gm%

Total number of cases	Age (Years)	Primi	G2	G3	G4	G5	G6
24	17-20	12	11	-	1	-	-
68	21-25	26	26	14	2	-	-
35	26-30	5	12	14	3	1	-
10	>30	1	4	-	1	1	3
137		44	53	28	7	2	3

Maximum patients clustered in the 21-25 year age group. They were mostly primis or second gravida. Beyond 30 years, women were of a higher gravida. As age increased the parity also increased.

17.5% of cases were teenagers.

They clustered round the primi and second gravida.

In my study early marriage, early pregnancy, frequent pregnancies with short interdelivery intervals were all factors that increased the incidence and prevalence of anaemia in the population of North Chennai.

TABLE - 2
AGE / GESTATIONAL AGE

Total Number of Cases	Age (Years)	Trimester		
		I	II	III
24	17-20	0	2	22
68	21-25	5	3	60
35	25-30	3	1	31
10	> 30	0	1	9
137		8	7	122

It was noticed in my study group that despite whichever age groups the anaemic patient belonged to, awareness was so poor, as most of them came to our hospital only in the III trimester or when they had symptoms. Most of the patients were labourers who had to earn a living even during pregnancy. They preferred to work, rather than to come for regular antenatal checkups and get admitted for treatment of anaemia unless it was very severe.

TABLE – 3
AGE / HAEMOGLOBIN

Total Number of Cases	Age (Years)	Haemoglobin	
		≤ 7 gm/dL	7.1 to 9
24	17-20	5	19
68	21-25	13	55
35	26-30	4	31
10	>30	3	7
137		25	112
		18.248%	81.7518%
		Severe Anaemia	Moderate Anaemia

18.24% of patients had severe anaemia and 81.75% had moderate anaemia in the group of patients with Hb≤ 9gm/dl.

TABLE 4
HAEMOGLOBIN DISTRIBUTION OF THE 1000 CASES

Total Number of Cases	Hb Conc. ≤ 7gm %	7.1to9gm%	9.1 to 10gm%	10.5 to 11gm%	Above 11 gm%
1000	25	112	786	40	37
	2.5%	11.2%	78.6%	4%	3.7%

Of the total 1000 patients – maximum patients had Hb between 9.1 to 10gm / dl (78.6%).

2.5% had severe anaemia

11.2% had moderate anaemia

78.6% had mild anaemia.

Patients with mild anaemia never even considered themselves to require treatment. Neither did they come for regular antenatal check up, nor were they compliant with oral iron therapy.

TABLE 5
GRAVIDA / HB

Total Number of Cases	Gravida	Haemoglobin	
		≤ 7 gm/dL	7.1 to 9gm/dL
44	Primi	4	40
53	G2	12	41
28	G3	3	25
7	G4	-	7
2	G5	1	1
3	G6	2	1

There were 48 G3 patients in the 1000 population studied.

Of these 25 were 7.1 to 9gm /dL

3 were ≤ 7gm /dL.

And 18 were 9.1 to 10 gm / dL.

Only 2 were > 10.5gm / dL.

Out of the 940 primis and gravida 2 only 97 had severe to moderate anaemia.

All patients who were fourth gravida and above suffered from moderate to severe anaemia.

Increasing parity does increase the incidence and severity of anaemia in the population of North Chennai.

The adoption of family planning was poor in North Chennai. Few patients came 4-6 weeks after delivery for CuT insertion. The acceptance of oral pills was also not very high. Most patients came as third or fourth gravida in the first or second trimester for MTP! This category included women with previous 2 LSCS who were totally unaware of the risk of MTP and those of another pregnancy. Hence we are strengthening our family welfare programmes and outreach services to tackle this group of patients.

TABLE 6

GESTATIONAL AGE / Hb

GA Trimester	Hb		Total
	≤ 7gm%	7.1 to 9gm%	
I	2	6	8
II	5	2	7
III	18	104	122
	25	112	137

Most of the patients with moderate and severe anaemia came for their first antenatal check-up only in the third trimester.

TABLE 7
HB / PERIPHERAL SMEAR

Total Number of Cases with Hb ≤ 9gm/dL	Peripheral Smear	
	Microcytic hypochromic	Normocytic hypochromic
137	95	42
	69.34%	30.656%

There were no cases of macrocytic anaemia. Maximum cases had microcytic hypochromic smear and showed drastic improvement with iron medication.

Nutritional deficiency was the major cause of anaemia in North Chennai. All the patients in the study group were unaware of food items rich in iron. As they came in the III trimester, they neither had iron medication, nor a chance to correct their dietary habits.

TABLE 8
BIRTH WEIGHT VS HAEMOGLOBIN

Birth weight ≥ 3Kg	≤ 7gm/dL	7.1 to 9gm /dL

≥ 3 kg	5	25
2.5 to 2.99 kg	5	45
2 to 2.499 kg	10	23
1.5 to 1.99 kg	3	15
1 to 1.499 kg	1	3

Low Birth Weight – A newborn whose weight is less than 2500 grams.

Very low birth weight - A newborn whose weight is less than 1500 grams

Extremely – Low birth weight – A new born whose weight is less than 1000 grams

Percentage Distribution of LBW in patients with Hb ≤ 9gm /dL (Moderate to severe anaemia)

In patients with moderate to severe anaemia 40.74% of babies were LBW.

Of the 40.74% LBW babies 7.27% were very low birth weight babies.

Patients who had initially Hb≤ 7gm% and came in I or II trimester (0.7% of the 1000 patients) had better outcomes. There was No incidence of LBW in these cases. Unbooked patients with severe anaemia in III trimester had more incidence of LBW.

This clearly shows the importance of regular antenatal checkups in improving the fetal outcome. Unfortunately in North Chennai it was a very small group (0.7%) who came early and enjoyed the privilege of a better fetal outcome.

TABLE 9
PATIENTS WITH Hb ≤ 9gm /dL Gravida Vs Birth weight

Gravida	Birth Weight	
	< 2.5 Kg.	2.5 kg and above
Primi	17 (38.63%)	27
Gravida 2	22 (42.30%)	30
Gravida 3	11 (40.74%)	16
Gravida 4	3 (50%)	3
Gravida 5	0	2
Gravida 6	1	2

As the gravida increases the percentage of LBW also increases.

The Gravida 5 and Gravida 6 patients were those where anaemia was detected and treated in II trimester.

TABLE 10

Gravida Vs Birth Weight in patients treated in I and II trimester and those who came in III Trimester

Weight Gravida in Kg.	**Treated early**		**Treated in III Trimester**	
	< 2.5	**2.5 & above**	**< 2.5**	**2.5 and above**
Primi	2	4	15	23
G2	2	2	20	28
G3	2	2	20	28
G3	0	3	11	13
G4	0	0	3	3
G5	0	1	0	1
G6	1	0	0	2

Gravida	**Treated early**		**Treated in III trimester**	
	<2.5 kg	**2.5 and above**	**<2.5Kg**	**2.5 and above**
Primi	2 (33.33%)	4	15 (39.47%)	23
Gravida 2	2 (50%)	2	20 (41.66%)	28
Gravida 3	0 (0%)	3	11 (45.83)	13
Gravida 4	0 (0%)	0	3 (50%)	3

Among primis the percentage of LBW was higher among the group which came in the III trimester.

All gravida 3 patients who were treated early had babies with birth weight above 2.5 kg.

Early treatment of anaemia does decrease the incidence of LBW.

TABLE 11
THE IMPACT OF INTER DELIVERY INTERVAL ON BIRTHWEIGHT

Inter delivery interval	Birth weight < 2.5 kg	2.5 kg and above	Total
< 2 years	15 (55.55%)	12 (44.44%)	27
$\geq$ 2 yrs	19 (36.538%)	33 (63.46%)	52

The percentage of LBW was definitely higher in women with inter delivery interval less than 2 years.

As age increased the parity also increased. Repeated pregnancies at short intervals along with a prolonged period of lactation in the population of North Chennai, put a serious strain on the iron-reserve. A normal healthy woman with adequate diet takes 2 years to replenish about 1000mg of iron lost during child birth and lactation.

TABLE 12
AGE VS BIRTH WEIGHT

Age	< 2.5 Kg	2.5 Kg and above
17-20 years	11 (47.82%)	12 (52.17%)
21-25	27 (46.278%)	40 (59.70%)
26-30	11 (32.35%)	23 (67.64%)
> 30 years	6 (60%)	4 (40%)

Maximum percentage of low birth weight fell into the > 30 years age group. The next were the teenage group.

Both teenagers and elderly women with anaemia were more prone for LBW. Teenagers have an added demand of 270mg iron for their natural growth. This extra requirement comes down to nil by the age of 21 years. Hence teenage pregnancies are more prone for anaemia.

TABLE 13
PRETERM Vs Hb

	≤ 7gm%	**7.1gm to 9gm%**
Number of Cases	3	19

A neonate born before 37 completed weeks (before 259th day)

TABLE 14
TERM / PRETERM Vs Hb

Number of Cases	≤7 gm/dL	7.1gm to 9gm / dL
Preterm	3 (12%)	19 (16.96%)
Term	22 (88%)	93 (83.04%)

16.96% of moderately anaemic patients and 12% of severely anaemic patients went for preterm labour.

Preterm babies had a higher risk of perinatal morbidity and mortality.

TABLE 15
MATURITY VS HAEMOGLOBIN

Number of Cases	≤9 gm/dL	9.1 to10gm/dl
Preterm	22 (16.05%)	29 (3.68%)

Patients with lower haemoglobin had higher incidence of preterm labour.

TABLE 16

AGE DISTRIBUTION OF ANAEMIC WOMEN WHO HAD PRETERM LABOUR

Age	No. of Cases	Percentage
15-20	4	18.18%
21-25	10	45.45%
26-30	6	27.27 %
> 30	2	9.09%

Of the 22 patients who had preterm labour.

3 were cases of preterm intrauterine deaths.

2 cases of early neonatal deaths.

1 was a case of multiple pregnancy.

TABLE 17
MODE OF DELIVERY IN ANAEMIC WOMEN WHO WENT FOR PRETERM LABOUR

Mode of Delivery	No. of Patients	Percentage
Vaginal	17	77.27%
Assisted Breech	1	4.54%
Outlet forceps	1	4.5%
LSCS	3	13.63%

APH

Of the 22 cases, there was

1 case of abruptioplancentae

1 case of placenta praevia type II B

HIV

There was 1 case with Hb 8.6gm% HIV +ve

The birth wt. was 1.8kg

It was a preterm baby

TABLE 18
BIRTHWEIGHT VS MATURITY

No. of Cases	**Birth Weight**

	< 2.5 kg	2.5 kg or above
Preterm	17	2
Percentage	89.47%	10.52%

89.47% of preterm babies were low birth weight. Lower the Hb more the likelihood of preterm labour. Prematurity increased the incidence of low birth weight, intrauterine death and early neonatal loss, thereby worsening the fetal outcome.

TABLE 19
MATERNAL OUTCOME

Hb Vs Maternal Cardiac Status

	Compensated	In failure	Total
Patients With Hb ≤ 7gm /dl	20 (80%)	5 (20%)	25
Patients with Hb>7gm /dL	112 (100%)	0	112
			137

Patients with moderate anaemia were not in failure.

Lower the Hb level the more likelihood of the patient being in failure. The 5 patients in this study who were in failure had Hb<5.6gm/dl. These patients were admitted immediately. The congestive cardiac failure was treated as mentioned earlier and patient kept hospitalised till delivery. Meanwhile the Hb status was improved by carefully transfusing packed cells (taking care to avoid volume over load) and giving the patients hematinics. Failure was uncommon when Hb was above 7gm/dl.

APH

Of the 137 patients with moderate and severe anaemia –

1 case of abruptio placentae

3cases of placenta praevia were observed.

ATONIC PPH

TABLE 20
ATONIC PPH Vs Hb

	Hb Level		
	≤ 7gm/dl	**7.1-9g/dl**	**≥ 9g/dl**
Total No. of Cases with Atonic PPH	1 (4%)	3 (2.6%)	2 (0.25%)
Total No. of Cases in each group	25	112	786

The lower the haemoglobin the greater the risk of atonic PPH.

One patient with atonic PPH had gone for acute inversion of the uterus. The same was repositioned and oxytocics were given to obtain hemostasis. A unit of packed cells was given to improve her general condition.

INFECTIONS

In the study group there was

1 case of diarrhoea

1 case of PUO

2 cases of LRI

1 case of UTI

1 case of skin lesions with dental carries

1 case of wound gaping in the puerperium.

MATERNAL MORTALITY

There was 1 maternal mortality in the study group.

That is 0.72% higher risk if Hb is less than 9gm/dl

0% if Hb > 9gm /dl

PERINATAL MORTALITY

2 (8%) deaths in patients with Hb $\leq$ 7gm /dl

3 (2.67%) deaths in patients with Hb 7.1 – 9gm / dl

A significant rise in Perinatal mortality with decreasing Hb.

STEPS TO REDUCE MATERNAL MORTALITY (ACTIONS FOR SAFE MOTHERHOOD).

A) Health Sector Actions.

- Basic antenatal, intranatal and post natal care. Risk assessment is a continued procedure throughout and is not only once.
- A skilled attendant should be present at every birth.

- Emergency obstetric care by field staff at the door step or preferably at the FRU.
- Good quality obstetric services at the referral centres.
- Prevention of unwanted pregnancy and unsafe abortion. All couples should have access to effective, client oriented and confidential family planning services.
- Frequent joint consultation amongst specialists in the management of anaemia in pregnancy.
- Maternal mortality conferences to evaluate the cause of death and the avoidable factors.
- Periodic refresher courses for continuing education of obstetricians, general practitioners, midwives and ancillary staff and to high light the preventable factors.

B) Community, Society and Family Actions

- These are essential to safe motherhood.
- Wide range of groups (Women's group), health care professionals, religious leaders and safe motherhood committees (regional, district) can help the women to obtain the essential obstetric care.

C) Health planners / Policy maker's action

- To organise community education, motivation and formation of safe motherhood committee at the local level.
- To strengthen the referral system for obstetric complications.
- To develop written management protocols for complications like congestive cardiac failure, atonic PPH, preterm labour.
- To improve standard and quality of care by organising refresher courses for health care personnel.
- Periodic audit of existing health care delivery system and to implement changes as needed.

D) Legislative and Policy Actions

- Girl children and adolescents should have good nutrition, education and economic opportunities.
- Barrier to the access of health care facilities should be removed.
- Policies should increase women's decision making power as regard to their own health and reproduction.
- Decentralisation of services to make them available to all the women.
- Post abortion care and treatment of anaemia in these women.
- Social inequalities and discrimination on grounds of gender, age and marital status are to be removed.

MEASURES HELPFUL IN REDUCING PERINATAL MORTALITY

- Pre-pregnancy health care and counselling
- To correct anaemia at this stage.

- To advice on birth spacing.
- Regular antenatal care with advice regarding health, diet and rest.
- Detection and correction of anaemia and prevention of pre-eclampsia.
- Screening of high risk patients, those of poor socioeconomic status, high parity, extremes of age, twins and their mandatory hospital delivery. Risk approach to RCH care is essential.
- Careful monitoring in labour.
- Skilled birth attendant – Minimise sepsis – clean hands, clean delivery surface and cut the cord clean.
- Provision of referral neonatal service to look after low birth weight and preterm babies.
- Health care education of the mother about the care of the new born- early and exclusive breast feeding.
- Educating the community to utilise family planning services and utilise the available MCH services. Family planning services can prevent unwanted pregnancies.
- Autopsy studies of all perinatal deaths.

- Continued study of perinatal mortality problems by demographic studies, regular clinically allied inter-departmental meeting and pathological research.

SUMMARY

- In this study of 1000 patients there were 2.5% severe, 11.2% moderate and 78.6% mildly anaemic patients.
- Most of the patients with moderate and severe anaemia came for the first time to our hospital only in the III trimester.
- Maximum number of patients fell in the mild anaemia group and of them most of patients had Hb between 9.1 and 10 gm /dl
- As the parity increases the Hb concentration decreases.
- 17.5% of cases were teenagers. Early marriage, frequent pregnancies at short intervals increased the incidence and severity of anaemia in the population.
- Most of the anaemic patients had microcytic hypochromic anaemia.
- The percentage of babies with birth weight less than 2.5kg was 40.74% among the moderate and severely anaemic patients.
- 28% of severely anaemic patients came for regular antenatal checkups and had no incidence of low birth weight.
- In unbooked anaemic patients as gravidity increased the percentage of low birth weight also increased.
- When the inter delivery interval was less than 2 years the incidence of low birth weight increased.

- There was poor acceptance of family planning measures especially when patients had children of same sex or when they wanted a male child.
- Anaemic women who belonged to the teenage group or > 30 year group had higher incidence of low birth weight.
- As Hb concentration decreases the incidence of prematurity increases. Prematurity increased the incidence of low birth weight, intrauterine death and early neonatal loss.
- Lower the Hb, more was the chance of congestive cardiac failure. The patients in this study had Hb < 5.8gm/dl.
- Lower the Hb the more was the risk of atonic PPH and infections.
- There was 1 Maternal mortality in the group with Hb < 9gm /dl and none in the group of mild anaemia.
- The perinatal mortality was 8% in severely anaemic patients and 2.67% in moderately anaemic patients, nearly a fourfold increase.

CONCLUSION

- The incidence of mild anaemia in North Chennai is very high. In most of them the pregnancy is uneventful. Nutritional anaemia and hookworm infestation were the most likely cause, as all patients who came early showed drastic improvement in maternal and fetal outcome with treatment of the same.

- Lower the haemoglobin, greater was the incidence of low birth weight, preterm labour and perinatal mortality. Early treatment did decrease the incidence of the above complications. But most of the patients paid their first visit only in the late third trimester. Women who had preterm labours had higher incidence of perinatal mortality.

- The study clearly showed that maternal morbidity like congestive cardiac failure, atonic PPH and infections and maternal mortality was higher when haemoglobin concentration fell.

- The people of North Chennai had no knowledge of the food items rich in iron and folic acid. The acceptance of family planning measures was poor. Repeated pregnancies with shortened inter-delivery intervals increased the severity of anaemia in this population. Poor hygiene, cooking taboos and fallacies played a major role in worsening the picture.

- This study emphasises the need for increased awareness, regular antenatal checkups, early detection and treatment of anaemia in the population of

North Chennai. It also emphasises the need to strengthen our out reach services and family welfare services.

BIBLIOGRAPHY

1) American college of Obstetricians and Gynaecologists. An update in Obstetrics and Gynaecology 2nd ed. 2000, p 82.

2) Andrews NC : Disorders of Iron Metabolism N. Engl. J. Med. 341-1986, 1999.

3) Bayouneu F. Subiran – Buisset C, Baka NE, et al – Iron Therapy in Iron deficiency anaemia in pregnancy. Intravenous route versus oral route. Am. J. Obstet Gynaecol. 186 : 518, 2002.

4) Murphy JF, O'Riordon J, New Combe RG, Coles EC, Pearson JF, relation of Haemoglobin Levels in first and second trimesters to outcome of pregnancy. Lancet 1986, 992-5.

5) Taylor DJ, Mollen C, Mc Dowgall N, Lind T. Effect of Iron supplements on serum ferritin levels during and after pregnancy. Br. J. Obstet. Gynaecol. 1982; 89 : 1011 – 17.

6) World Health Organisation : Nutritional Anaemias. WHO, Geneva, 1972.

7) WHO. Prevention and Management of Severe anaemia in pregnancy. Report of a Technical Working Group, Geneva 20-22 May 1991 Maternal Health and Safe Motherhood Programme WHO, Geneva, 1993.

8) Suenberg B. Absorption of Iron in pregnancy Acta Obstet & Gynaecology Second (Suppl) 1975, 48-. 1-108.

9) Thompson WG (1988). Comparison of tests for diagnosis of iron depletion in pregnancy. American Journal of obstetrics and gynaecology 159, 1132-1134.

10) Williams' Text Book of Obstetrics 22nd Edition, Iron Deficiency Anaemia.

11) Arias High Risk Pregnancy Anaemia Complicating Pregnancy.

12) Iron Stores in Pregnancy – Br. J. Haematology 1977. British Journal Obst. Gyne. 1991: 98-886.

13) Medical Disorders in Obstetrics Practice by Michael De Sweit.

14) Massay AC Med.Clin. Northan 1992, May. 76 : 549-66 studied the microcytic anaemia – diagnosis of iron deficiency anaemia.

15) Tenton V, Cavilli, Fisher J. Br. J. Haemalol 1977 Sept. 37. 145-9, studied the iron stores in pregnancy.

16) IVC metk, Andrelassy, K. Mann V. F Czes E.L., Suno Nouts I, Hematology (Budop) 1981, 14 : 307-13, Lewis G.I., Rowe DFBJ. Clin Practice 1986. Jon. 40-156.

17) Iron deficiency anaemia in pregnant women, diagnostic aspects studied byHarma K, Herrig T, Vlvik RJ, Tidssler Nor-Laegetoren Maoan N, Rusion V, Mono A. Isood S.K.

18) Clodfelter RL – Emerg Med.ClinNorth Am.1986,Feb4;59-74 study the peripheral smear of anaemia in pregnancy.

19) Hallberg. I Ryttinger, Solvell L (1666), side effects of oral Iron Therapy. Acta Medica Scandinavia 459, 3-16.

20) Bentley DP (1985), Iron Metabolism and Anaemia in pregnancy. Clinics in Haematology, 14, 613-628.

21) Letskey EA (1987) Anaemia in Obst. In Studd (ed) Progress in Obstetrics and Gynaecology pp 23-58. Edinburgh, Chrochil Livingstone.

22) Finch CA. Cokk JD (1984) Iron deficiency, Amercian Journal of Clinical Nutrition 39, 471-477.

PROFORMA

Name : Age : I.P.No. Serial No. :

Husband's Name Occupation and Income :

Address : Religion :

Date of Admission : Height:

Date of Discharge : Weight :

Present History :

H/o. Breathlessness

H/o Worm infestation

H/o. Chronic blood loss

H/o. Haemarrhoids

H/o. Malaria

MENSTRUAL HISTORY

H/o. Frequency of cycles, duration of flow

H/o. Passing blood clots

Last menstrual period

OBSTETRIC HISTORY

Gravida Para Live Abortion

Birth Spacing

LMP (Trimester)

EDD

Past Medical and Surgical History

Diet History

Physical Examination

- Stature
- Build
- Presence of Pallor
- Examination of Cardiovascular system
- Examination of Respiratory System
- Abdomen

INVESTIGATIONS

Hb gm/dl

Hematocrit

TRBC count

MCV MCH

MCHC

TWBC

DC

Platelet Count

Peripheral Blood Smear

T protein

Se Albumin

Motion ova

Cyst

ULTRASOUND

ASSOCIATED FACTORS

- PIH
- Abruption
- Infections

MOTHER

- Compensated / Failure
- Shock
- PPH
- Peripheral vein thrombosis
- Maternal Death

DELIVERY DETAILS

Term / Preterm

Vaginal / Caesarean

BABY

Alive / IUD

Term / Preterm

Birth weight

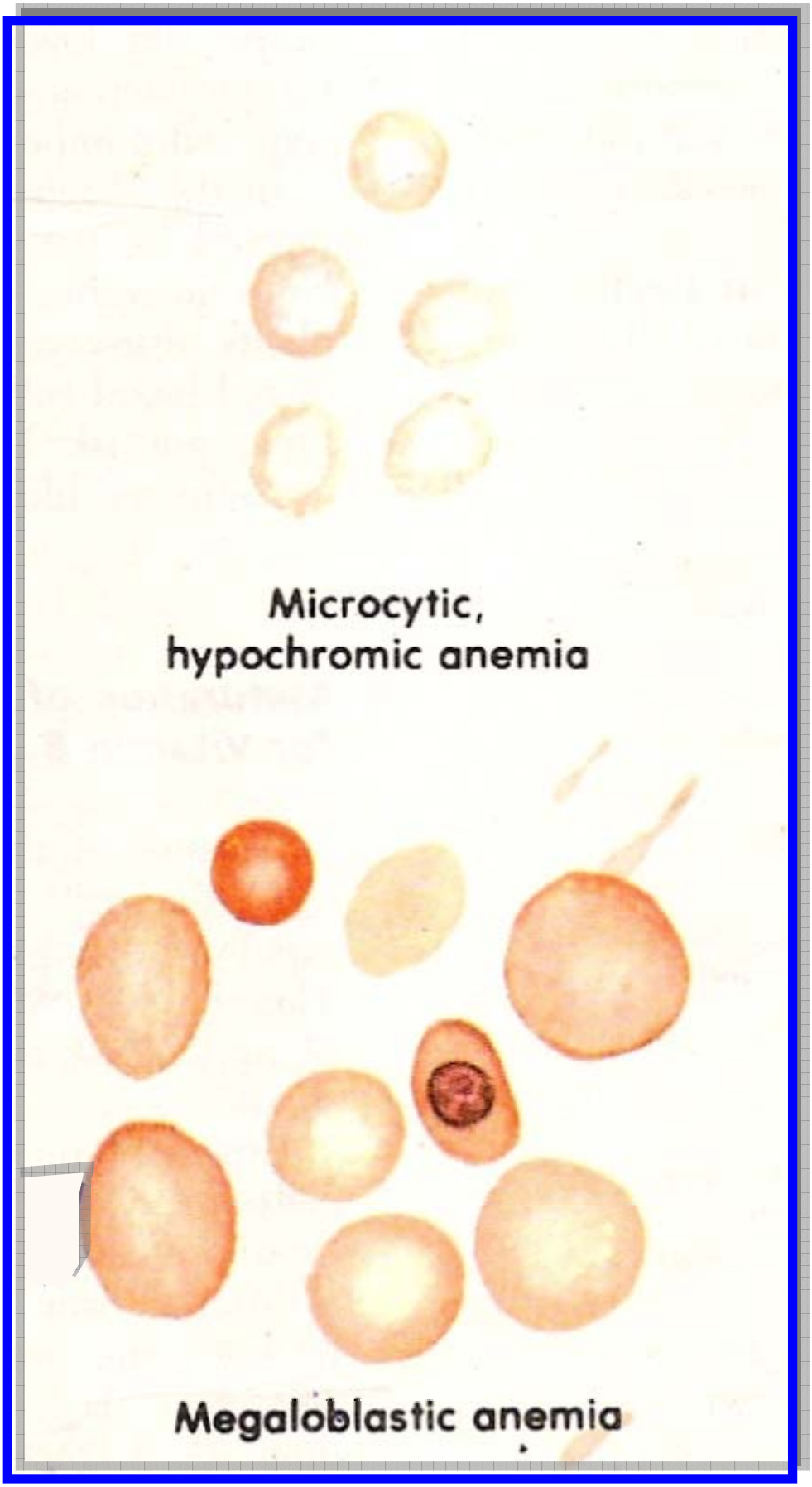
Apgar Scoring at 1 minute and 5 minutes

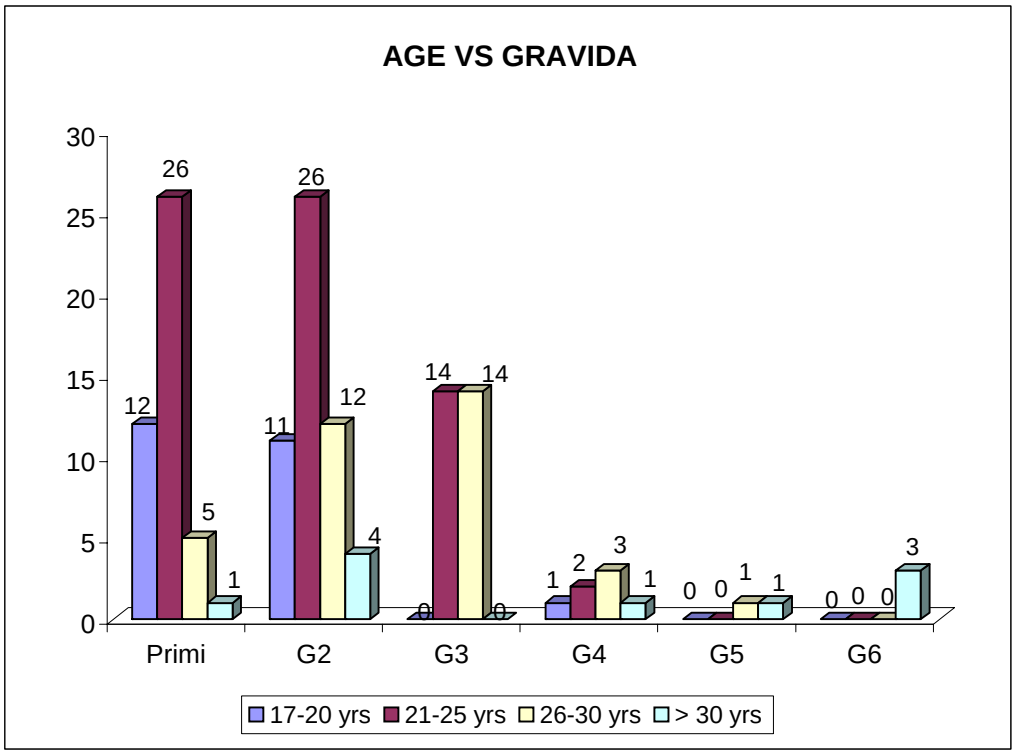
NEONATAL PERIOD

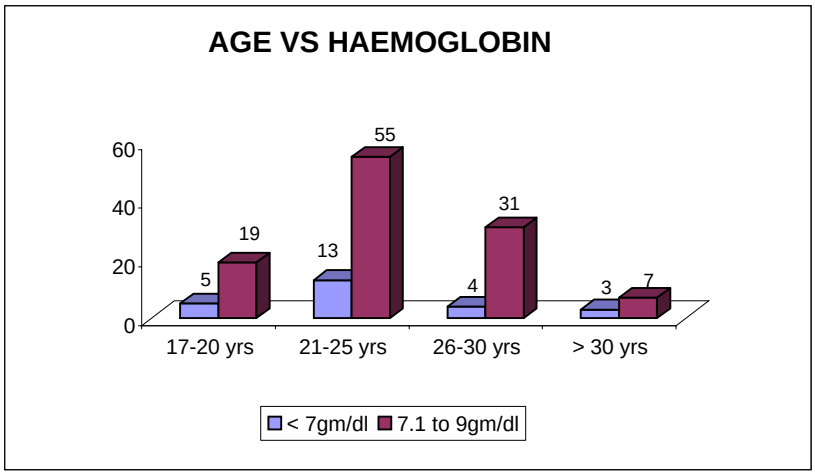
- Alterations of sensorium
- Sepsis
- Convulsions
- Jaundice
- Birth injury
- Neonatal death

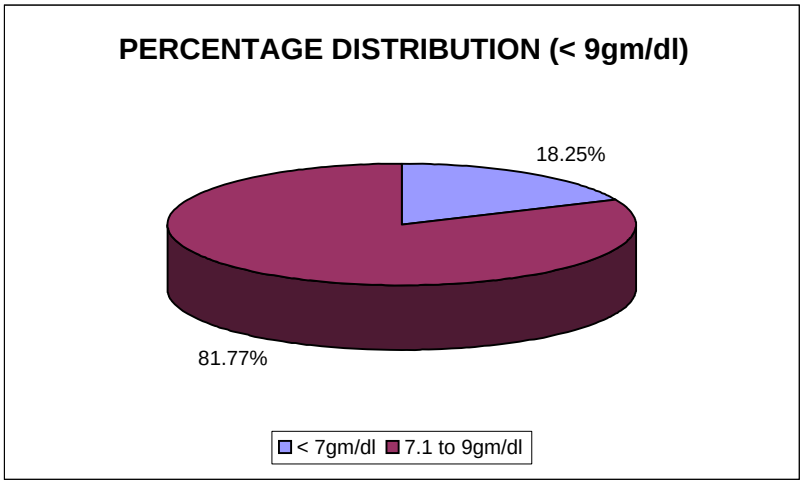
TREATMENT RECEIVED

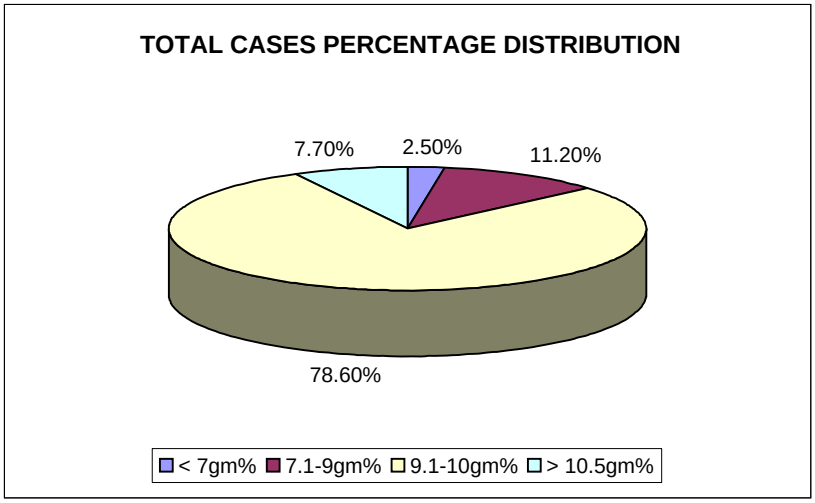
- Oral iron and folate tablets
- IV Iron – Imferon
- Packed cell transfusion
- Treatment of failure.

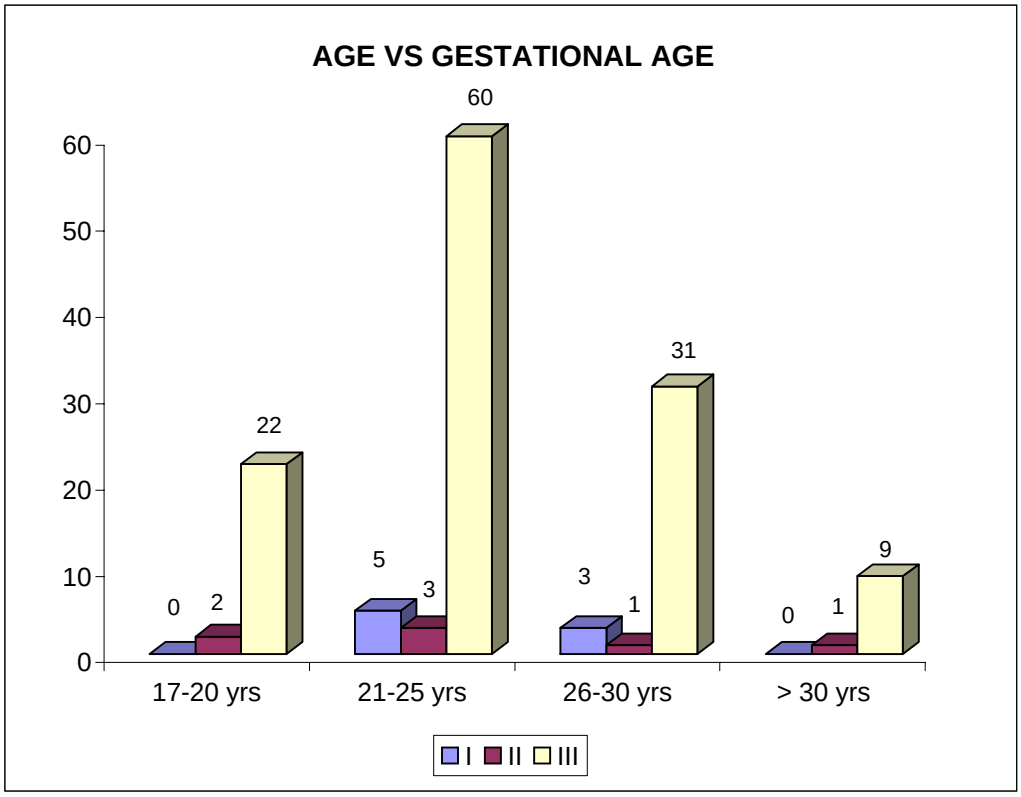

Microcytic,
hypochromic anemia
Megaloblastic anemia

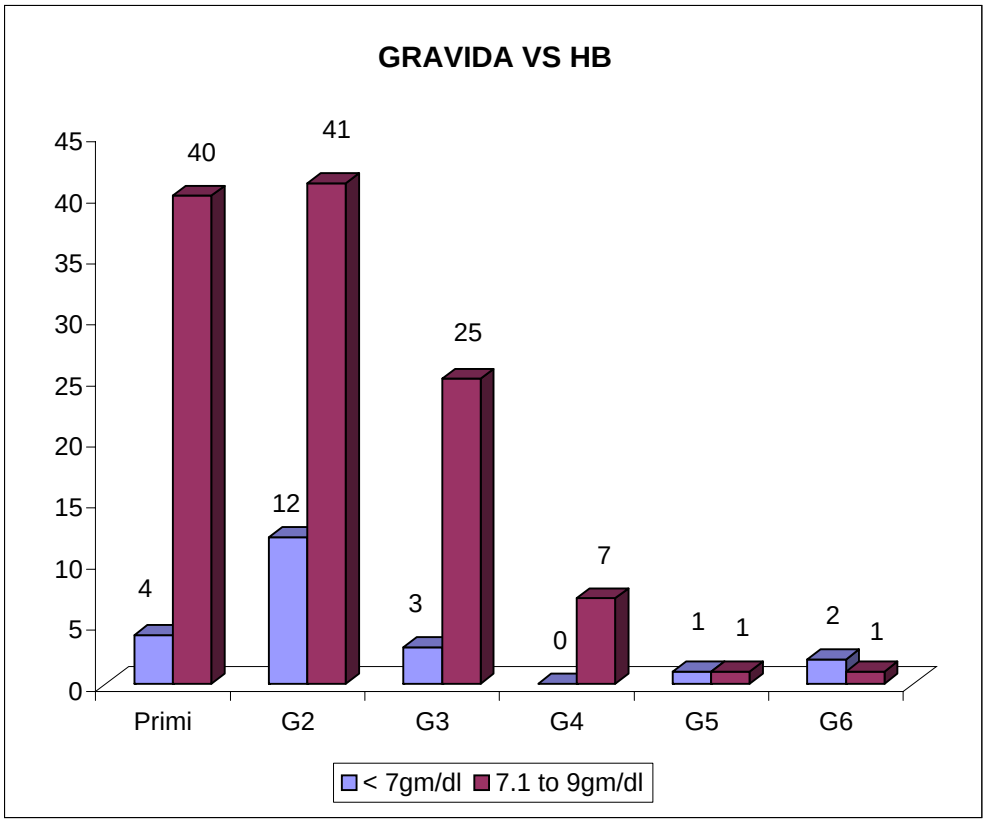

AGE VS GRAVIDA
30
25
20
15
10
5
0
12
26
5
1
11
26
12
4
0
14
14
0
1
2
3
1
0
0
1
1
0
0
0
3
Primi
G2
G3
G4
G5
G6
17-20 yrs
21-25 yrs
26-30 yrs
> 30 yrs

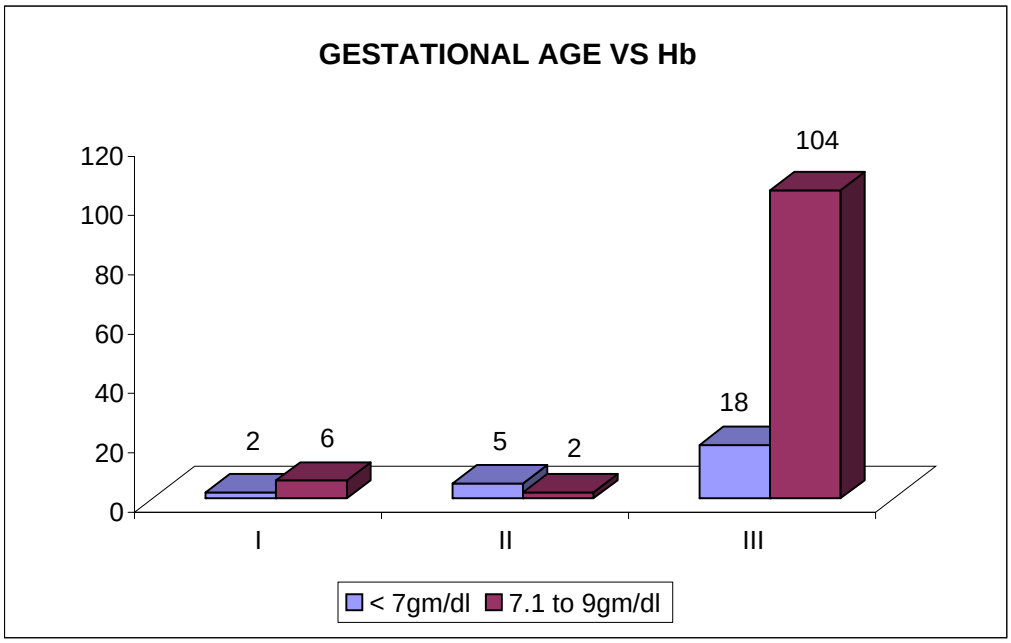

AGE VS HAEMOGLOBIN
60
40
20
0
5
19
13
55
4
31
3
7
17-20 yrs
21-25 yrs
26-30 yrs
> 30 yrs
< 7gm/dl
7.1 to 9gm/dl

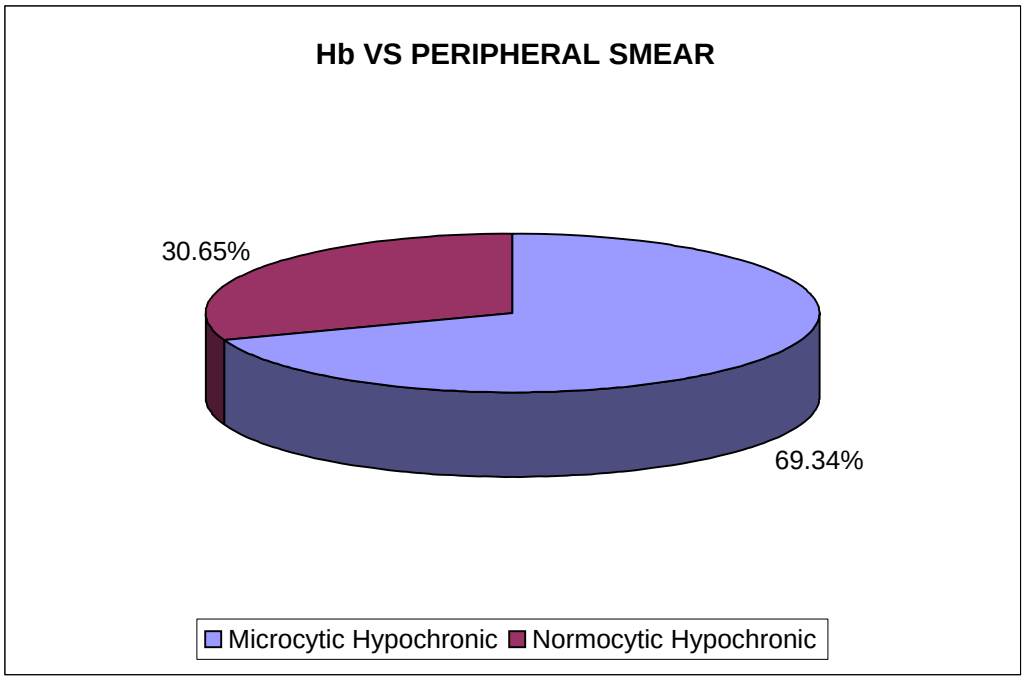

PERCENTAGE DISTRIBUTION (< 9gm/dl)
18.25%
81.77%
< 7gm/dl
7.1 to 9gm/dl

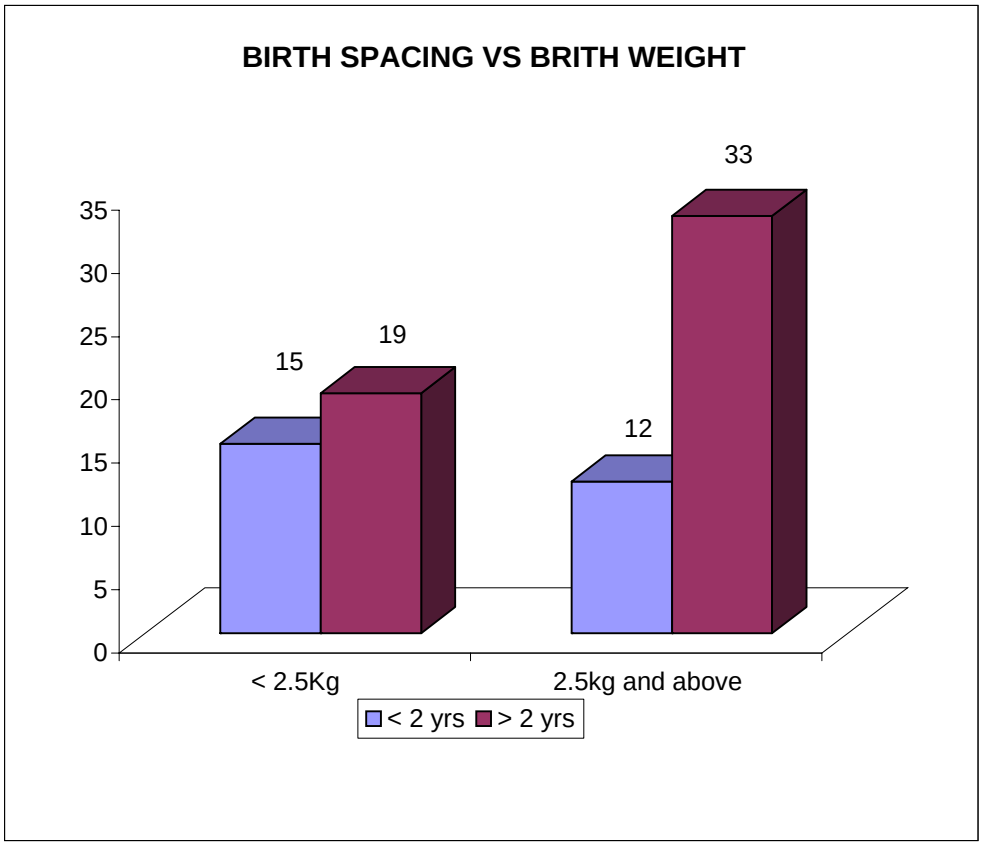

TOTAL CASES PERCENTAGE DISTRIBUTION
7.70%
2.50%
11.20%
78.60%
< 7gm%
7.1-9gm%
9.1-10gm%
> 10.5gm%

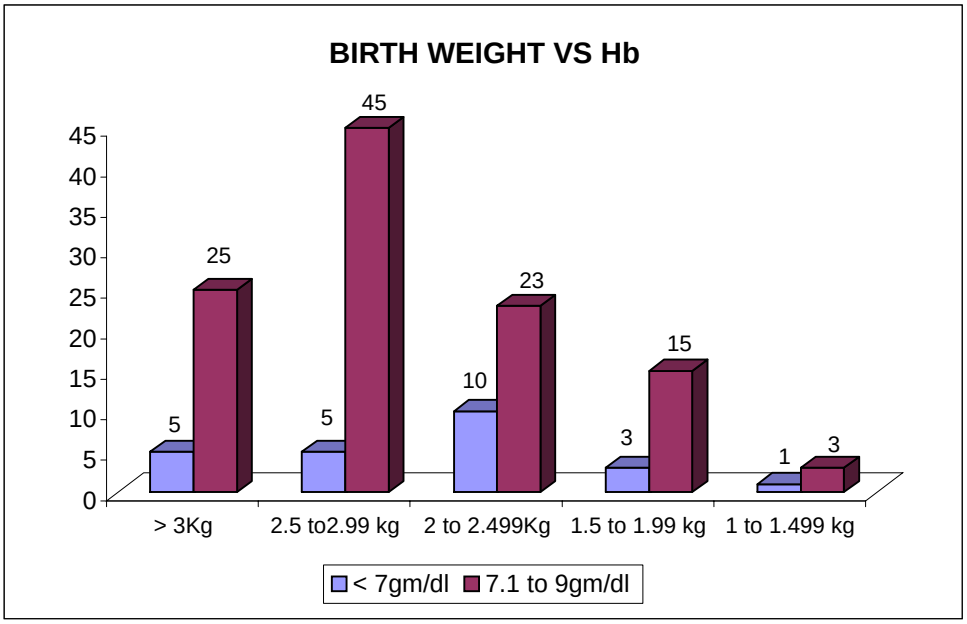

AGE VS GESTATIONAL AGE
60
50
40
30
20
10
0
0
2
22
5
3
60
3
1
31
0
1
9
17-20 yrs
21-25 yrs
26-30 yrs
> 30 yrs
I
II
III

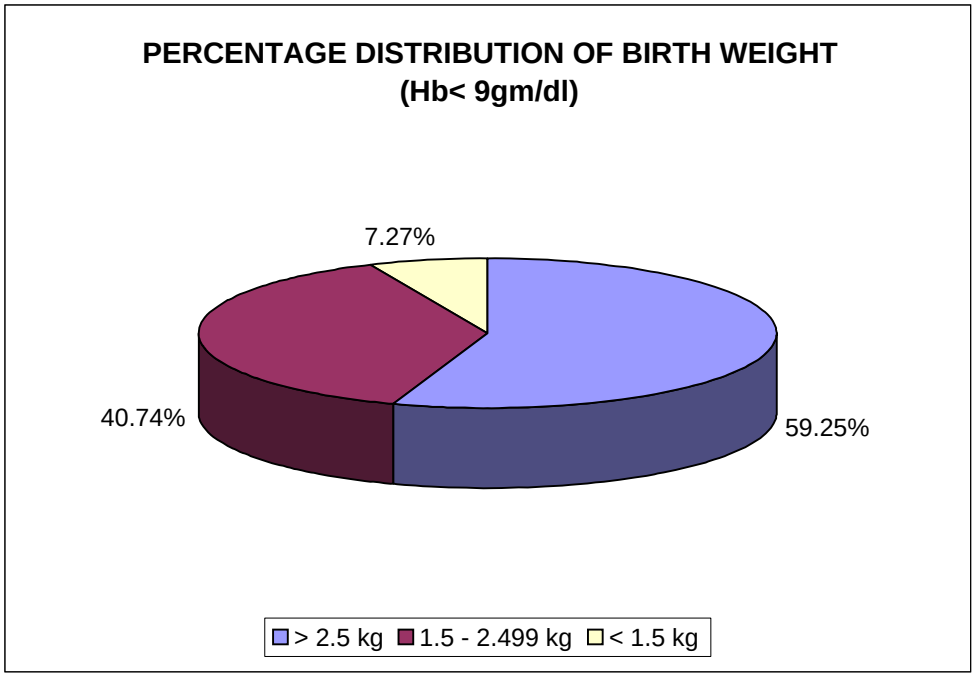

GRAVIDA VS HB
45
40
35
30
25
20
15
10
5
0
4
40
12
41
3
25
0
7
1
1
2
1
Primi
G2
G3
G4
G5
G6
< 7gm/dl
7.1 to 9gm/dl

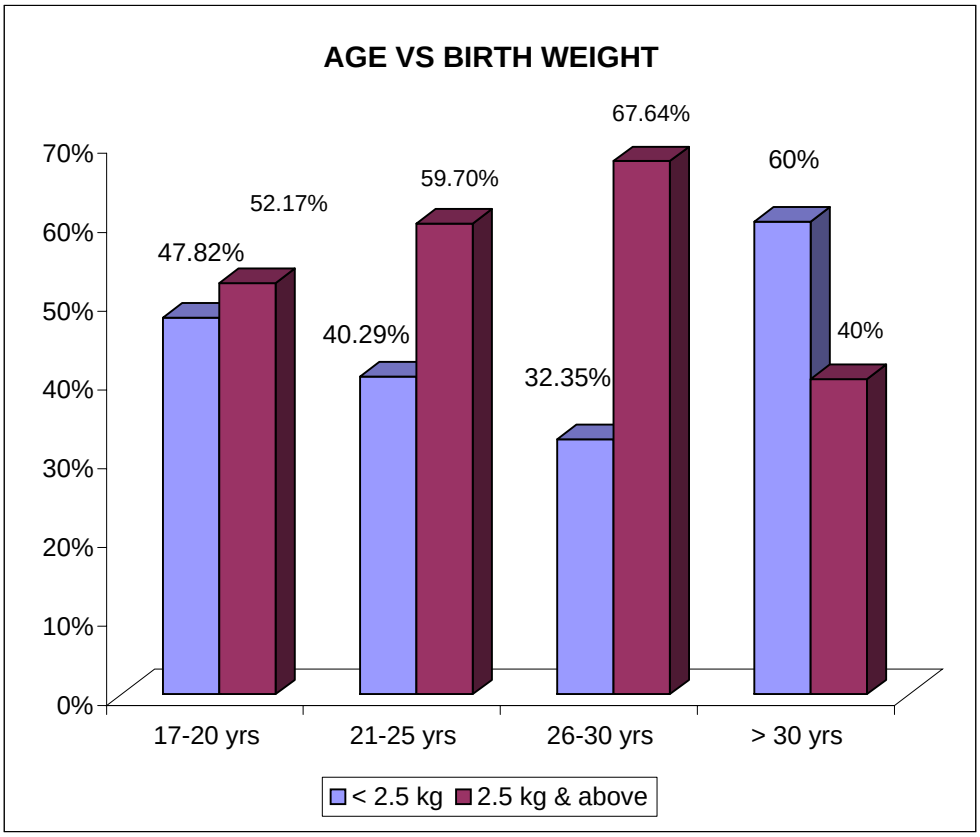

GESTATIONAL AGE VS Hb
120
100
80
60
40
20
0
2
6
5
2
18
104
I
II
III
< 7gm/dl
7.1 to 9gm/dl

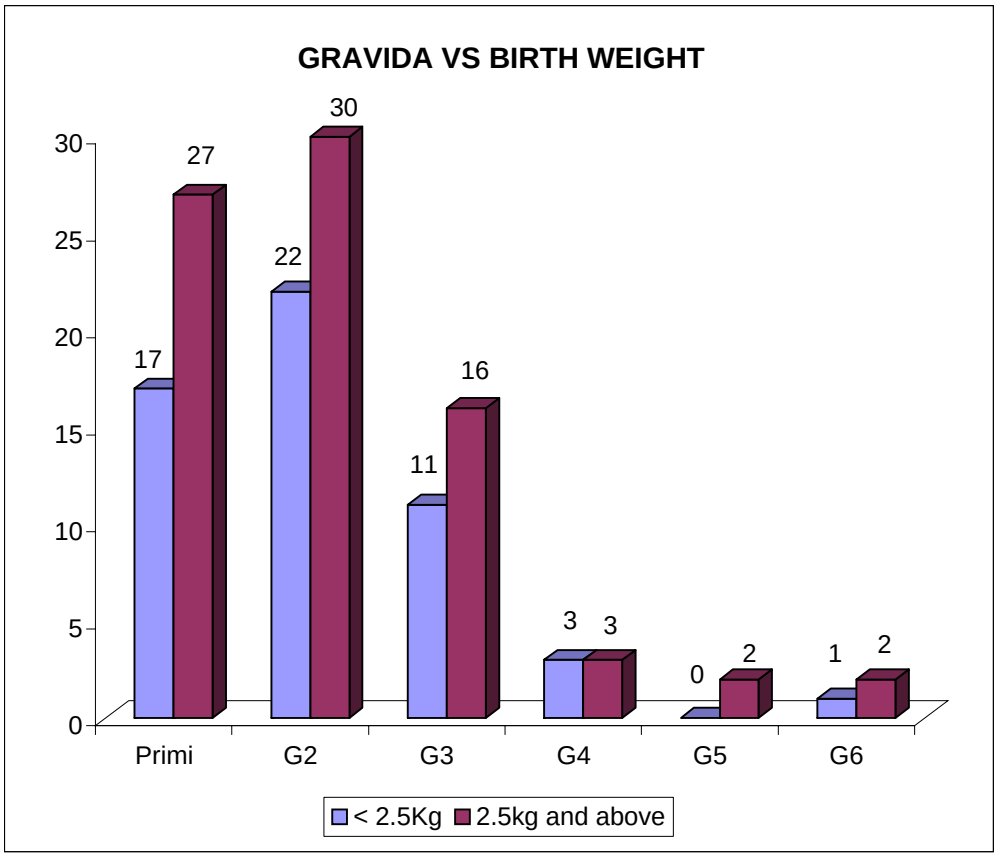

Hb VS PERIPHERAL SMEAR
30.65%
69.34%
Microcytic Hypochronic
Normocytic Hypochronic

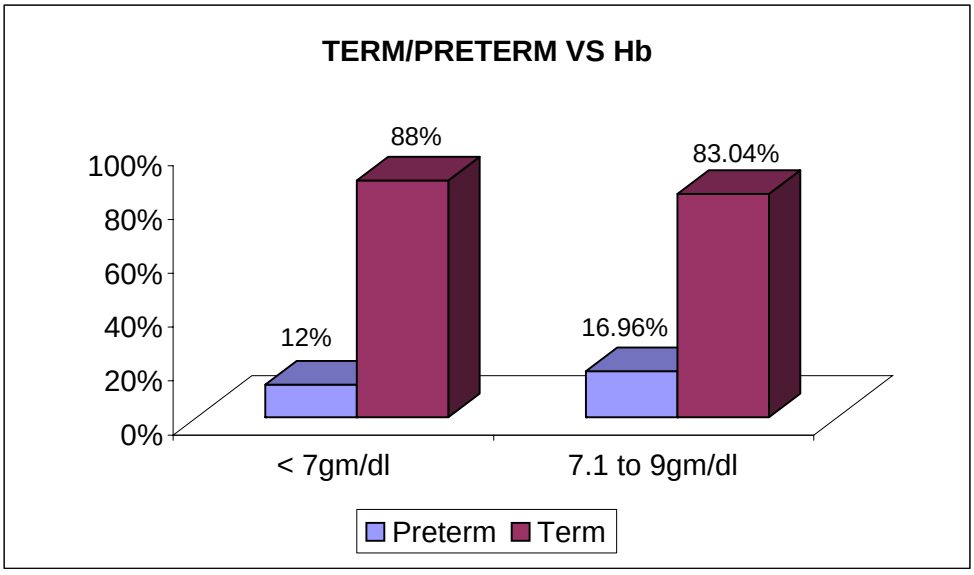

BIRTH SPACING VS BRITH WEIGHT
35
30
25
20
15
10
5
0
15
19
12
33
< 2.5Kg
2.5kg and above
< 2 yrs
> 2 yrs

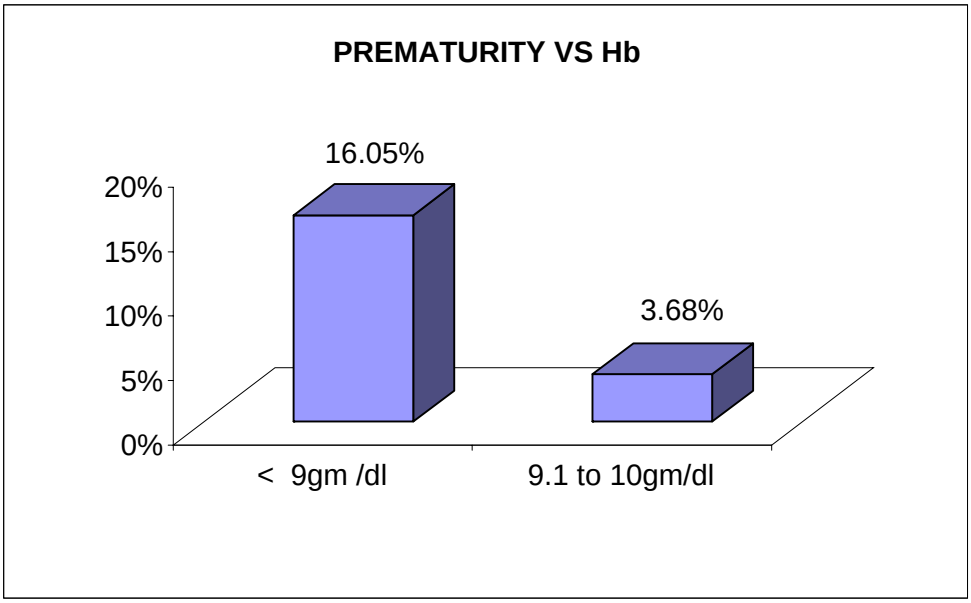

BIRTH WEIGHT VS Hb
45
40
35
30
25
20
15
10
5
0
5
25
5
45
10
23
3
15
1
3
> 3Kg
2.5 to2.99 kg
2 to 2.499Kg
1.5 to 1.99 kg
1 to 1.499 kg
< 7gm/dl
7.1 to 9gm/dl

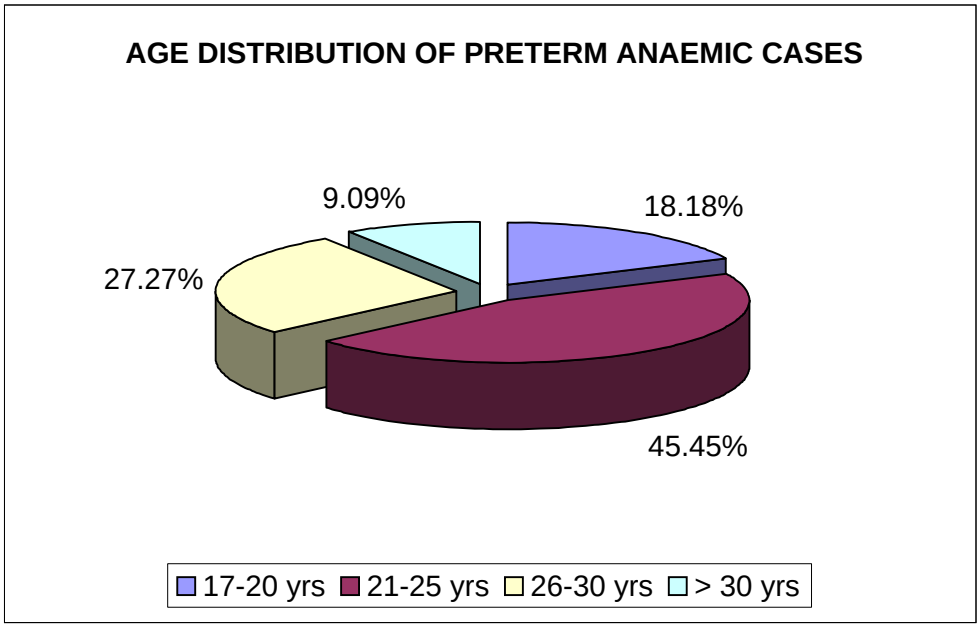

PERCENTAGE DISTRIBUTION OF BIRTH WEIGHT
(Hb< 9gm/dl)
7.27%
40.74%
59.25%
> 2.5 kg
1.5 - 2.499 kg
< 1.5 kg

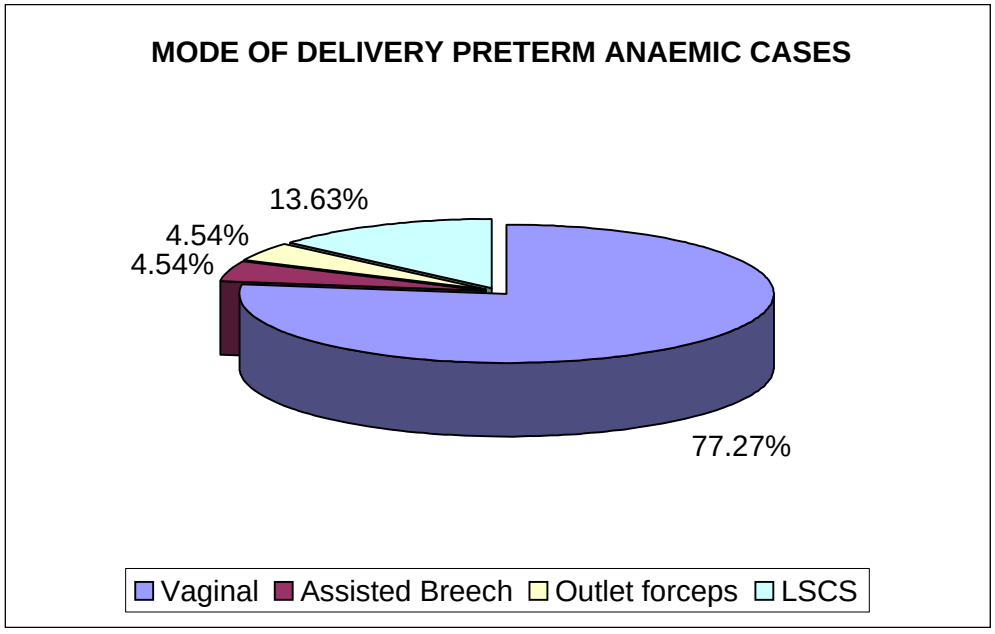

AGE VS BIRTH WEIGHT
70%
60%
50%
40%
30%
20%
10%
0%
47.82%
52.17%
40.29%
59.70%
32.35%
67.64%
60%
40%
17-20 yrs
21-25 yrs
26-30 yrs
> 30 yrs
< 2.5 kg
2.5 kg & above

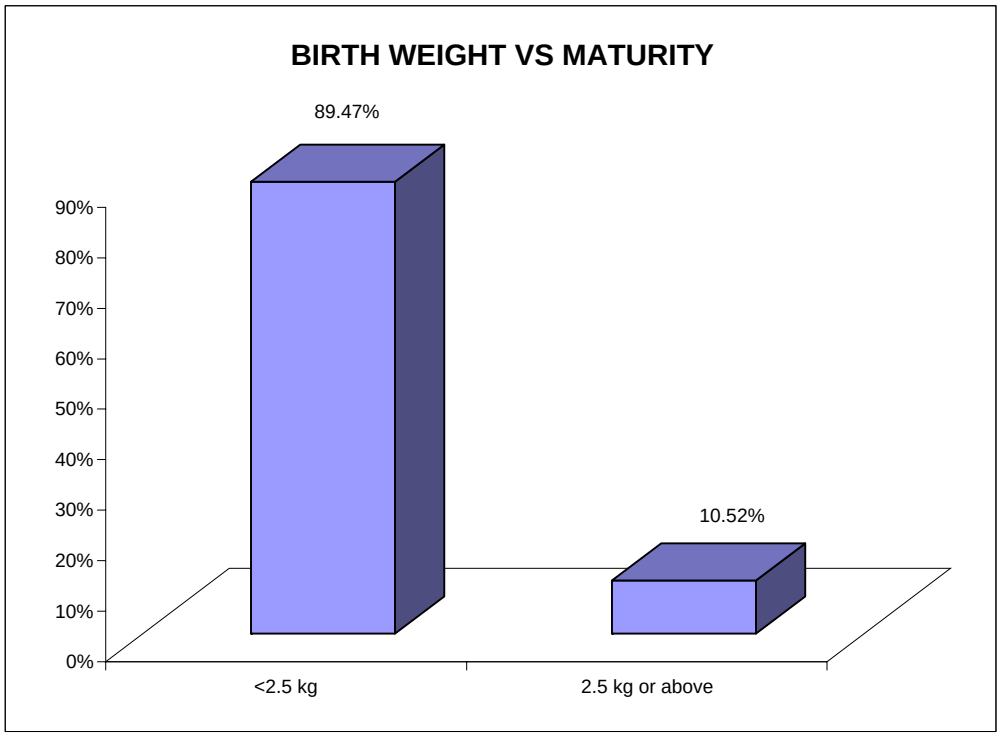

GRAVIDA VS BIRTH WEIGHT
30
25
20
15
10
5
0
17
27
22
30
11
16
3
3
0
2
1
2
Primi
G2
G3
G4
G5
G6
< 2.5Kg
2.5kg and above

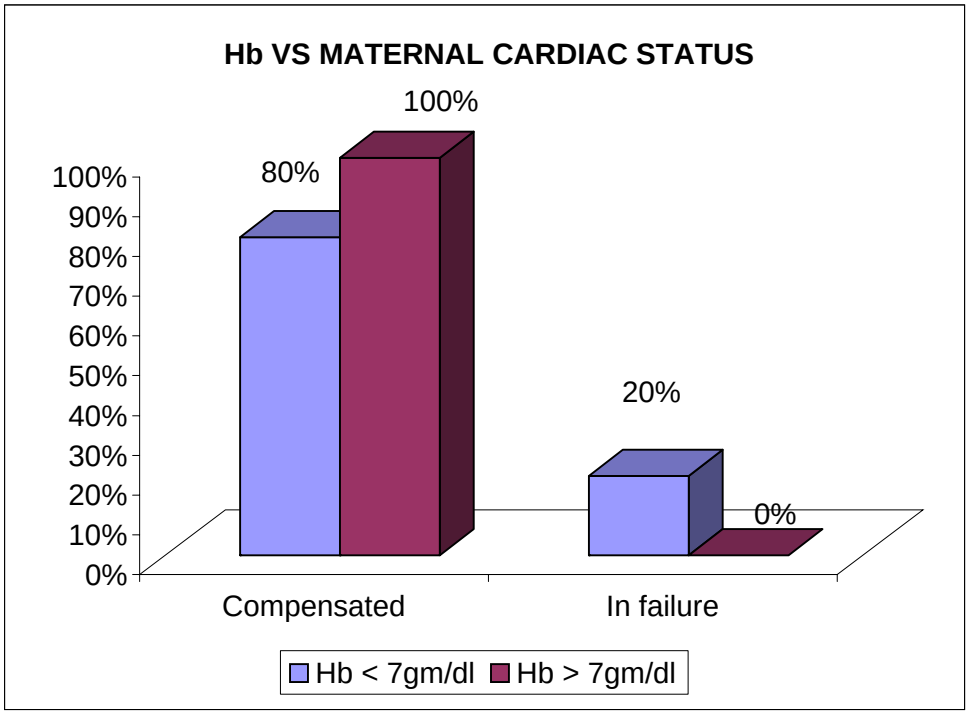

TERM/PRETERM VS Hb
100%
80%
60%
40%
20%
0%
88%
12%
83.04%
16.96%
< 7gm/dl
7.1 to 9gm/dl
Preterm
Term


PREMATURITY VS Hb
20%
15%
10%
5%
0%
16.05%
3.68%
< 9gm /dl
9.1 to 10gm/dl


AGE DISTRIBUTION OF PRETERM ANAEMIC CASES
9.09%
18.18%
27.27%
45.45%
17-20 yrs
21-25 yrs
26-30 yrs
> 30 yrs


MODE OF DELIVERY PRETERM ANAEMIC CASES
13.63%
4.54%
4.54%
77.27%
Vaginal
Assisted Breech
Outlet forceps
LSCS


BIRTH WEIGHT VS MATURITY
89.47%
10.52%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
<2.5 kg
2.5 kg or above


Hb VS MATERNAL CARDIAC STATUS
100%
80%
20%
0%
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
Compensated
In failure
Hb < 7gm/dl
Hb > 7gm/dl

MASTER CHART

Sl. No.	IP No.	Age	Gravida	First Vist at	Hb	Birth spacing (Years)	Associated factors	Maternal Morbidity	Maternal mortality	Mode of Delivery	Fetal				Neontal Deaths
											Alive / IUD	Term / Preterm	Birth Weight	Apgar Score/10 for 1' & 5'	
1	11265	26	2	III	8.6	10	-	-	-	LSCS	Alive	Term	2.6	6, 9	-
2	12369	24	Primi	III	8.8	-	-	-	-	L-Naturale	Alive	Term	2.9	8, 9	-
3	11612	21	2	III	8	1	Post molar preg.	-	-	L-Naturale	Alive	Term	2.6	6, 10	-
4	11729	35	6	III	7	6	-	-	-	L-Naturale	Alive	Term	2.4	6, 8	-
5	11585	45	Primi	III	8.6	-	Breach	-	-	LSCS	Alive	Term	2.4	8, 10	-
6	11575	20	Primi	III	8.2	-	-	-	-	LSCS	Alive	Term	2.5	6, 8	-
7	11110	27	5	II	6	3	-	-	-	L-Naturale	Alive	Term	3.25	7, 8	-
8	11821	30	3	III	8.2	4	-	-	-	L-Naturale	Alive	Term	2.9	7, 8	-
9	11894	18	Primi	III	8.6	-	-	-	-	L-Naturale	Alive	Term	2.9	8, 9	-
10	11527	20	3	III	8.8	3	Prev. LSCS	-	-	LSCS	Alive	Term	2.4	7,8	-
11	11370	23	2	III	8.8	2	Prev. LSCS	-	-	LSCS	Alive	Term	2.5	6, 8	-
12	12225	23	Primi	I	4	-	-	-	-	L-Naturale	Alive	Term	2.6	6, 8	-
13	12226	35	2	III	8.6	6	-	-	-	L-Naturale	Alive	Term	2.9	5, 6	-
14	10435	35	2	III	7.6	1	Multiple pregnancy	-	-	L-Naturale	Alive	Preterm	1.7, 1.75	6,8	-
15	9523	26	Primi	I	7.4	-	HbsAg +ve, Rh -ve	-	-	LSCS	Alive	Term	2,5	4,7	-
16	11554	35	2	III	8.6	3	Central Placenta Praevia	-	-	LSCS	Alive	Term	2.75	6, 9	-
17	11479	22	Primi	III	8.6	-	-	-	-	Outlet forceps	Alive	Term	3.25	6, 8	-
18	11573	35	6	III	8	2	-	-	-	L-Naturale	Alive	Term	3.5	6, 8	-
19	11478	24	Primi	III	8.4	-	-	-	-	L-Naturale	Alive	Term	2.3	6, 8	-

20	11456	20	2	III	8.6	1 3/4	Multiple pregnancy	-	-	L-Naturale	Alive	Term	2.5, 2	6,8	-
21	11448	23	Primi	III	8	-	-	-	-	L-Naturale	Alive	Preterm	2.25	8, 10	-
22	11427	25	2	III	6.8	6		-	-	L-Naturale	Alive	Term	3	8,9	-
23	11137	19	2	III	8.2	1	-	-	-	LSCS	Alive	Term	1.8	3, 8	-
24	11297	20	2	II	8.6	1½	-	Diarrhoea	-	L-Naturale	Alive	Term	2.4	6,8	-
25	11277	28	2	III	6.2	4		-	-	L-Naturale	Alive	Term	2	6, 8	-
26	11026	20	Primi	III	8	-	Breech	-	-	Assisted Breech	Alive	Preterm	1.95	2, 6	-
27	10622	21	Primi	III	4.8	-	-	Congestive failure	-	L-Naturale	Alive	Preterm	1.5	6, 8	+
28	10865	20	2	III	6.8	2	Placenta Previa IIB	-	-	LSCS	Alive	Preterm	2.65	7, 9	-
29	11120	23	2	III	8.8	4	-	-	-	L-Naturale	Alive	Term	2.75	5, 7	-
30	11231	18	Primi	III	8	-	-	-	-	L-Naturale	IUD	Preterm	1.25	-	-
31	11218	23	3	III	8.8	3	Prev. LSCS	-	-	LSCS	Alive	Term	3.1	5, 8	-
32	10688	30	Primi	III	8	-		-	-	LSCS	Alive	Preterm	1.7	5, 8	-
33	11155	30	Primi	III	8.6	-	Breech	-	-	LSCS	Alive	Term	3.25	6, 8	-
34	11226	22	2	III	8.8	1½	-	-	-	L-Naturale	Alive	Term	3.25	6, 8	-
35	11242	37	2	III	7.6	1	-	-	-	L-Naturale	Alive	Term	2.3	5, 8	-
36	11547	18	2	III	8.6	1½		-	-	L-Naturale	Alive	Term	2.3	6, 8	-
37	12156	21	Primi	III	8.6	-	HIV +ve	-	-	L-Naturale	Alive	Preterm	1.8	6, 7	-
38	12136	25	3	III	6.8	-	-	-	-	L-Naturale	Alive	Term	3	6, 8	-
39	11904	20	Primi	III	6.2	-	-	-	-	L-Naturale	Alive	Term	2.1	6, 8	-
40	11876	19	Primi	III	8	-	-	Atonic PPH	-	L-Naturale	Alive	Term	2.75	6, 8	-
41	12158	23	Primi	I	-	-	-	-	-	L-Naturale	Alive	Term	2.25	4, 6	-
42	12241	21	Primi	III	8.3	-	-	-	-	L-Naturale	Alive	Term	3.25	8, 9	-
43	12335	20	Primi	III	8.6	-	-	-	-	L-Naturale	Alive	Preterm	1.8	4, 8	-
44	12336	22	Primi	III	8.4	-	-	-	-	L-Naturale	Alive	Term	3.3	5, 8	-
45	11961	23	Primi	III	7.6	-	-	Wound gaping	-	LSCS	Alive	Term	2.5	5, 8	-
46	12549	20	2	III	5.5	1½	Placenta Previa IIB	-	-	LSCS	Alive	Term	2.5	8, 9	-
47	12137	29	2	III	8.8	1	Prev. LSCS	-	-	LSCS	Alive	Term	3.7	5, 8	-

48	12152	25	Primi	III	8	-	HbsAg +ve	-	-	LSCS	Alive	Term	3.35	8, 9	-
49	12710	23	Primi	III	8	-	-	--	-	L-Naturale	Alive	Term	2.5	6, 8	-
50	12596	26	Primi	III	8.8	-	-	-	-	L-Naturale	Alive	Term	2.5	8, 9	-
51	11897	23	Primi	III	8.8	-	-	UTI	-	L-Naturale	Alive	Preterm	1.45	6,7	-
52	11842	24	2	III	6	1½	Prev. LSCS, Heart Disease	-	-	L-Naturale	Alive	Term	2.25	5, 8	-
53	12402	25	2	III	7.2	2	-	-	-	L-Naturale	Alive	Preterm	2.5	6, 8	-
54	12325	28	3	III	8.4	4	-	-	-	L-Naturale	Alive	Term	3.5	8, 10	-
55	12324	27	3	III	8.4	4	-	-	-	L-Naturale	Alive	Term	3.25	9, 10	-
56	11781	19	Primi	II	5.6	-	-	-	-	L-Naturale	Alive	Term	2.3	6, 8	-
57	10887	29	3	III	5.6	3	multiple pregnancy	-	-	L-Naturale	Alive	Term	1.9, 2	7, 8	-
58	11834	28	2	III	7.4	2 3/4	multiple pregnancy	-	-	L-Naturale	Alive	Term	1.8, 1.9	7, 9	-
59	11835	18	Primi	III	8.8	-	-	-	-	L-Naturale	Alive	Term	2.75	6,8	-
60	11551	28	4	III	7.6	2	-	-	-	L-Naturale	Alive	Term	2.7	6, 8	-
61	11366	26	4	III	8.8	1	-	Gr.I Abruption	-	L-Naturale	Alive	Preterm	1	4, 6	-
62	11465	32	6	III	6	1	-	Congestive failure	-	L-Naturale	Alive	Term	2.4	6, 8	-
63	11097	21	2	III	7	2	-	-	-	L-Naturale	Alive	Term	2.9	7,8	-
64	12400	22	2	III	8.6	1	-	-	-	L-Naturale	Alive	Preterm	1.55	6, 8	-
65	12541	19	Primi	III	8	-	-	-	-	LSCS	Alive	Term	2.6	8, 9	-
66	12958	21	Primi	III	8.6	-	-	-	-	L-Naturale	Alive	Term	2.4	8, 9	-
67	12949	21	Primi	III	8.6	-	-	-	-	L-Naturale	Alive	Term	3.5	6, 8	-
68	11543	25	3	III	5	3	-	-	-	L-Naturale	Alive	Term	2.9	7, 9	-
69	11535	26	2	III	8.4	4	-	-	-	L-Naturale	Alive	Term	2.5	8, 10	-
70	11848	22	3	III	8.6	1½	-	-	-	LSCS	Alive	Term	2.8	6, 8	-
71	8343	23	Primi	III	8.6	-	-	-	-	Outlet forceps	Alive	Term	2.9	5, 6	-
72	11930	30	3	III	8	1	-	-	-	L-Naturale	Alive	Term	1.75	7, 9	-
73	12035	22	3	I	8.8	1	-	-	-	L-Naturale	Alive	Term	2.6	6,8	-
74	12159	35	5	III	8	1	-	-	-	L-Naturale	Alive	Term	2.5	6, 8	-
75	12104	27	3	III	7.8	1½	-	-	-	L-Naturale	Alive	Term	3.25	6, 8	-
76	12237	25	3	III	8	1½	-	-	-	L-Naturale	Alive	Term	2.2	8, 10	
77	13048	25	3	III	7.6	2	-	-	-	L-Naturale	IUD	Preterm	1	-	-

78	11969	24	Primi	III	8.6	-	Rh -ve	-	-	L-Naturale	Alive	Term	2.6	6, 8	-
79	12193	18	Primi	III	8	-	-	-	-	L-Naturale	Alive	Term	2.7	4, 8	-
80	11847	23	Primi	I	8.8	-	-	-	-	L-Naturale	Alive	Term	2.6	6, 8	-
81	11555	25	Primi	III	6	-	-	Atonic PPH	-	L-Naturale	Alive	Term	2.3	8, 9	-
82	11508	33	3	III	8.8	11	-	-	-	L-Naturale	Alive	Preterm	2.1	5,9	-
83	11873	21	2	III	8	1	-	-	-	L-Naturale	Alive	Term	1.5	3, 5	-
84	11877	24	4	III	8.4	2½	PUO	-	-	L-Naturale	Alive	Preterm	2.1	6, 9	-
85	11960	22	2	III	8.8	1	-	-	-	Outlet forceps	Alive	Term	2.6	6, 8	-
86	11844	22	2	III	8.8	3	-	-	-	L-Naturale	Alive	Term	1.9	6,8	-
87	11901	30	2	III	8	1½	Schizophrenia	-	-	L-Naturale	Alive	Term	2.75	6,8	-
88	12331	28	2	III	8.6	4	-	-	-	L-Naturale	Alive	Term	3	8,9	-
89	12477	28	3	III	8	1	Breech	-	-	Assisted Breech	Alive	Term	2.4	4, 6	-
90	12633	26	3	I	8.2	4	-	-	-	L-Naturale	Alive	Term	2.6	6, 8	-
91	12699	20	2	III	8.2	1		-	-	L-Naturale	Alive	Term	2.8	6, 8	-
92	12729	30	2	III	8	5	-	-	-	L-Naturale	Alive	Term	2	6, 8	-
93	12784	19	2	III	8.8	1	-	-	-	L-Naturale	Alive	Term	2.8	8, 9	-
94	12228	27	3	III	8	3	-	-	-	L-Naturale	IUD	Preterm	1	-	-
95	13092	21	4	III	8.6	1	-	Atonic PPH	-	L-Naturale	Alive	Term	2.3	6, 8	-
96	12821	26	3	III	7.6	2	-	-	-	L-Naturale	Alive	Preterm	1.6	6,8	-
97	12973	22	2	I	4.8	2	-	Congestive failure	-	L-Naturale	Alive	Term	2.3	6, 8	-
98	12837	25	Primi	III	8	-	Rh -ve	-	-	L-Naturale	Alive	Term	2.75	6, 8	-
99	12790	30	2	III	8.8	1½	-	-	-	L-Naturale	Alive	Term	2.2	6, 8	-
100	12789	23	3	III	8	4	-	-	-	L-Naturale	Alive	Preterm	1.75	4, 8	-
101	12517	25	3	III	8.8	2	Prev. 2 LSCS	-	-	LSCS	Alive	Term	3.5	8, 9	-
102	12500	25	2	III	7	2	-	-	-	L-Naturale	Alive	Term	2.25	6,7	-
103	10478	27	3	III	8	1½	-	-	-	L-Naturale	Alive	Preterm	1.2	4,6	+
104	10933	23	2	III	7.6	2		-	-	L-Naturale	Alive	Term	1.25	4, 5	+
105	10977	21	3	III	8.6	1 3/4	-	-	-	L-Naturale	Alive	Term	2,25	6, 8	+
106	10724	23	2	III	8.8	2	-	-	-	LSCS	Alive	Term	3.1	7, 8	-
107	10805	25	3	III	8.8	3	-	-	-	L-Naturale	Alive	Term	2.7	5, 8	-
108	10631	22	2	III	8.8	4	-	-	-	L-Naturale	Alive	Term	2.75	5, 8	-

109	11095	23	2	III	8.8	3	-	-	-	L-Naturale	Alive	Term	2.4	8, 9	-
110	10737	28	2	III	8.8	2	-	-	-	L-Naturale	Alive	Term	2.75	5, 8	-
111	10744	25	Primi	III	8.2	-	-	-	-	L-Naturale	Alive	Term	3	6,8	-
112	10585	30	4	III	8.6	3		-	-	L-Naturale	Alive	Term	2.9	5, 8	-
113	10609	30	2	III	8	3	-	LRI	-	L-Naturale	Alive	Term	3.3	6, 8	-
114	10755	21	2	III	7	1½	-	-	-	L-Naturale	Alive	Term	2.5	6, 8	-
115	10756	28	3	III	8.6	2	Prev. LSCS	-	-	VBAC	Alive	Term	3.2	6, 8	-
116	10766	20	Primi	III	8.8	-		-	-	L-Naturale	Alive	Term	2	6,8	-
117	10779	24	2	III	8	-	-	-	-	L-Naturale	Alive	Term	2.5	8, 10	-
118	9582	22	2	III	8	5	-	LRI	-	LSCS	Alive	Term	1.9	6, 8	-
119	9763	20	2	III	5.8	1	-	-	-	L-Naturale	Alive	Term	1.9	6, 8	-
120	9664	25	4	III	8.8	1½	-	-	-	L-Naturale	Alive	Term	2.5	6, 8	-
121	9691	30	3	III	8.4	1	-	-	-	L-Naturale	Alive	Term	2.75	6, 8	-
122	9940	18	2	III	7.8	1½	-	-	-	L-Naturale	Alive	Term	2.6	6,8	-
123	9553	23	Primi	III	8.8	-	-	-	-	LSCS	Alive	Term	3.1	6,8	-
124	10681	23	2	III	8.6	-	-	-	-	L-Naturale	Alive	Term	3.75	6, 8	-
125	9367	20	2	III	8.8	1	-	-	-	LSCS	Alive	Term	2.8	3, 4	+
126	10210	29	2	I	8.8	1	-	-	-	L-Naturale	Alive	Term	3	6, 8	-
127	9962	24	2	III	8.6	2½	-	-	-	L-Naturale	Alive	Term	2.3	6, 9	-
128	9992	24	Primi	II	8.8	-	-	-	-	L-Naturale	Alive	Term	2.5	6, 7	-
129	7564	24	2	III	6.6	2	-	-	-	Outlet forceps	Alive	Preterm	2	6, 7	-
130	10228	22	Primi	III	8.8	-	-	-	-	L-Naturale	Alive	Term	3.25	7, 8	-
131	9937	23	2	III	8.6	2	-	Atonic PPH	-	L-Naturale	Alive	Term	2.5	6,8	-
132	9983	22	2	III	8	1½	-	-	-	L-Naturale	Alive	Preterm	1.8	6,8	-
133	9741	23	3	III	8.8	5	-	-	-	L-Naturale	Alive	Term	2.1	6, 8	-
134	10168	22	Primi	III	8	-	-	Dental Carries Skin Lesions	+	L-Naturale	Alive	Term	2	8, 10	-
135	12865	28	3	III	7	5	-	-	-	L-Naturale	Alive	Term	3.3	7, 9	-
136	13054	24	Primi	8.2	-	-	-	-	-	L-Naturale	Alive	Term	3.25	8, 10	-
137	13078	25	3	8.8	3	-	-	-	-	L-Naturale	Alive	Term	3	6, 8	-